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**Case Report**

# A case of recurrent achalasia cardia with sigmoid esophagus managed laparoscopically

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## ABSTRACT

Recurrences of symptoms after the surgery for achalasia cardia are not uncommon. There are several causes of recurrences but the early recurrences are thought to be secondary either to incomplete myotomy or late recurrences due to fibrosis after the myotomy or megaesophagus. A laparoscopic approach is a standard method because of the obvious benefits for the patients. The extent of the myotomy and addition of fundoplication are debatable issue in the management of achalasia cardia but evidence suggests that some kind of fundoplication would be necessary after the complete division of lower esophageal sphincter. We present our experience in a case of recurrent achalasia, secondary to incomplete myotomy managed laparoscopically by extended myotomy and a Dors fundoplication.

**Keywords:** Recurrences of symptoms, Laparoscopic approach, Achalasia

## INTRODUCTION

Achalasia is a rare disease of the esophagus characterized by the inability of the lower esophageal sphincter (LES) to relax and causes varied esophageal contractions. The incidence of achalasia is 1.63/100,000.

Patients with achalasia typically present with dysphagia for liquids more than solids, regurgitation of undigested food, heartburn, halitosis and chest pain. It has been reported that dysphagia occurs in over 90% of achalasia patients.[1] Dysphagia can be largely categorized as a motility disorder (achalasia, oropharyngeal dysfunction). Given the nonspecific clinical presentation of achalasia, this may mistake symptoms of achalasia for gastroesophageal reflux disease (GERD) or pseudo achalasia due to malignancy.

The etiology of achalasia is largely unknown. It has been postulated that achalasia is linked to a viral or autoimmune inflammatory response, leading to selective degeneration of inhibitory neurons of the myenteric plexus.[3] Patients suspected of achalasia undergo diagnostic examinations such as oesophagogastroduodenoscopy (OGD), barium swallow and high-resolution chest computed tomography (CT).[4] Ultimately, a definitive diagnosis is made with manometry as a gold standard investigation.[5]

As the disease progresses, dilation of the esophagus worsens and can resemble a sigmoidal shape. In late-stage achalasia, patients present with a sigmoid esophagus which is defined as a dilation of the distal esophagus to more than 10 cm in diameter and/or one that takes a tortuous course through the chest towards the gastroesophageal junction.

Treatment for late-stage achalasia with severely dilated and sigmoidal shaped esophagus has been controversial. Some have argued that a laparoscopic Heller's myotomy with Dor fundoplication, as used to treat early-stage achalasia, have promising outcomes for late-stage achalasia as well. Others have argued a more aggressive approach of esophagectomy may be needed to bring substantial relief.[6]

**Table 1: Severity of the disease.**

| Stages | Description |
|---|---|
| **Stage 0** | Esophageal width of 4 cm or less |
| **Stage I** | Esophageal width of between 4 and 6 cm |
| **Stage II** | Esophageal width of greater than 6 cm |
| **Stage III** | Marked dilation of the distal esophagus >10 cm in diameter, tortuous course, angulation ± axis deviation |

The severity of the disease is staged by evaluating the dilatation degree of esophageal body in centimeters by measuring the maximum esophageal width from standard posteroanterior projection esophagograms.[7]

Manometric findings of esophageal body contractions along with high LES basal pressure and inadequate relaxation of LES.[8] Chest radiography can show a widened mediastinum, a mediastinal air-fluid level, or absence of a gastric air bubble.[9] CT show findings of grossly dilated fluid-filled esophagus, tracheobronchial tree compression and the frequently seen lung changes as sequela of aspiration like consolidation, fibrotic patch, ground glass or nodular opacities. A diameter of >6 cm and axis deviation are regarded to represent a sigmoid esophagus, the distal esophagus is kinked toward the left, outside of the esophageal axis.

Although complete absence of peristalsis in the esophageal body has been proposed as the major abnormality in achalasia, the evidence indicates achalasia as a primary disorder of the LES. This is based on 24-hour outpatient esophageal motility monitoring which shows that even in advanced diseases up to 5% of contractions can be peristaltic.[10]

**Table 2 Chicago classification.[10]**

| Motility disorder | Defining criteria |
|---|---|
| **Type 1** | Elevated IRP, 100% failed peristalsis |
| **Type 2** | Elevated IRP, 100% failed peristalsis with PEP in > 20% swallows |
| **Type 3** | Elevated IRP, 100% failed peristalsis and >20% swallow with spasm |
| **EGJ outflow obstruction** | Elevated IRP, normal peristalsis |
| **Hypercontractile esophagus** | >20% hypercontractile swallow |
| **Distal esophageal spasm** | Normal IRP >20% swallows with spasm |

## CASE REPORT

A 60-year-old male admitted in the surgery department with near absolute dysphagia for 2 months and regurgitation of food particles.

He gave a previous history of similar complaints 40 years ago for which he was operated via a upper midline incision but operative details are not unavailable.

In the past 20 years he had difficulty in swallowing which progressively worsened since the past 2 months in which he developed absolute dysphagia to both solids and liquids.

He had multiple episodes of vomiting post meals with food particles present.

### *Investigations*

Routine blood investigations were done and were within normal limits.

An initial barium swallow was done which was suggestive of tortuous distal esophageal dilatation (Figure 1a and b). followed by an upper GI scopey were in the scope could not be negotiated past the gastroesophageal junction. The finding included food particles present in the esophagus with massive dilatation in the distal oesophagus.

An esophageal manometry was attempted but unsuccessful as the guide wire could not be negotiated past the gastroesophageal junction.

A repeat manometry was attempted after keeping the patient on a liquid diet for 3 days but was unsuccessful.

An HRCT suggestive of gross dilatation of thoracic esophagus with air fluid levels with no mucosal involvement being present. Severe scaring in the distal esophagus was present.

A diagnosis of achalasia cardia was made after this thorough workup.

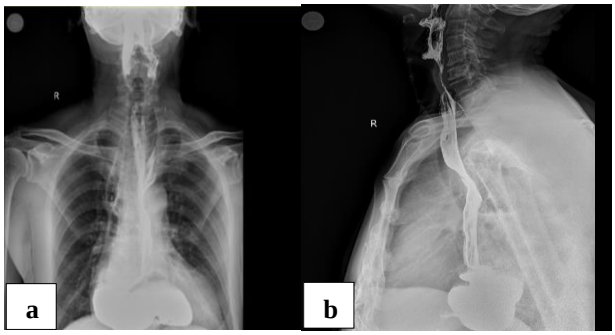


**Figure 1: Pre-operative barium swallow (a) anterior view, and (b) lateral view.**

### *Operative findings*

The port positions were same as for laparoscopic hiatus hernia repair. The liver was completely adherent to the diaphragm and the distal end of the oesophagus. The surgery was difficult because of loss of planes, distorted

anatomy, excessive scarring and severe adhesions of the OG junction to the left lobe of the liver. After adhesiolysis around the hiatus area. The esophagus was mobilized circumferentially. The posterior vagus nerve was identified, the anterior vagus nerve was seen proximally in the mediastinum to the left of the previous myotomy and was lifted away to complete the myotomy, excessive care was taken not to injure the vagi. The proximal myotomy was extended to 6 cm and the lower limit of the cardio myotomy was extended up to 3 cm on the stomach to completely divide the lower esophageal sphincter. Myotomy was performed. intra-operative endoscopy was done, the scope was negotiated in the stomach with some difficulty due to the tortuous distal esophagus and excessive scarring around the GE junction.

Completeness of the myotomy was confirmed on table endoscopy and at the same time air leak test was done to rule out any mucosal perforation. The Dor fundoplication was done after dividing the short gastric vessels (Figure 2). The Surgery lasted for 120 minutes.

There were no peri-operative complications.

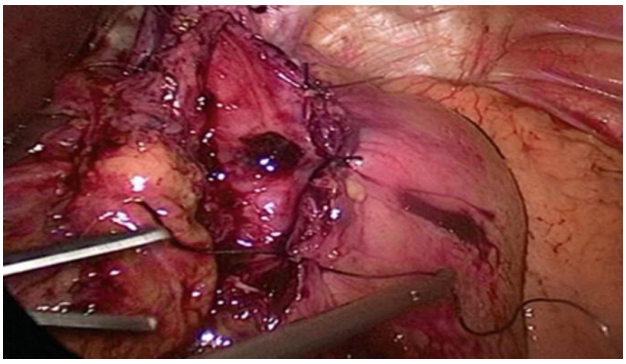

**Figure 2: Intraoperative image of Dor's fundoplication.**

He was fed liquids on first postoperative day.

His symptoms improved significantly and a postoperative barium meal done at 2 weeks post-surgery showed significant reduction in the esophageal diameter and free passage of barium in the stomach (Figure 3a and b).

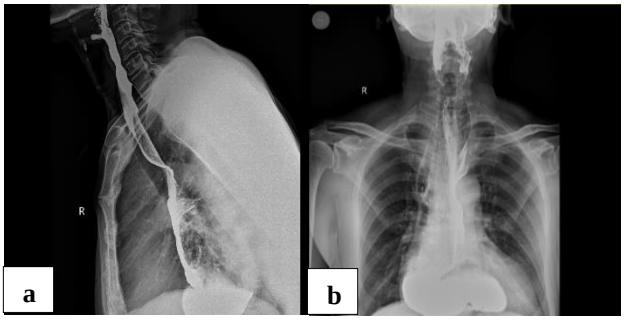


**Figure 3: Post-operative barium swallow (a) LAT, and (b) AP.**

## DISCUSSION

The most common causes of these recurrences are incomplete and/or inadequate myotomy, scarring, megaesophagus prior to surgery, constricting fundoplication and gastroesophageal reflux.

It is advised that the completeness of the myotomy should be checked intraoperatively by endoscopy or manometry.

The extent of the myotomy and addition of fundoplication is a debatable issue in the management of achalasia cardia but the evidence suggest that some form of fundoplication would be necessary after the complete division of lower esophageal sphincter.

The most common post-operative complication for myotomy is GERD. This is seen in 40% of post-operative patients. To mitigate this complaint an anterior Dor's fundoplication or posterior Toupet is needed. The use of a 360° nissen's is considered contraindicated due to the increase in resistance against an aperistaltic esophageal body.

A retrospective cohort taken 10 years post Heller myotomy demonstrated more than 50% of patients had reflux symptoms without fundoplication

In a randomised clinical trial 7% of patients undergoing Dor fundoplication with Heller's myotomy had abdnormal 24-Hour PH monitoring Heller-Dor gives better results than Heller myotomy alone.[11]

Other debatable issues in these procedures is the mobilization of the esophagus in the mediastinum, the need for anti-reflux procedure and extension of myotomy in either direction is easily done laparoscopically.

A thoracic approach can also be tried but the mediastinal approach is difficult with a greater morbidity and it also requires a double lumen tube for anesthesia, lateral decubitus position, difficult in ergonomic position to access the esophagus, a need for a chest drain post operatively, postoperative pain, longer operative time, longer hospital stay and a higher incidence of persistent dysphagia and secondary gastroesophageal reflux postoperatively.

## CONCLUSION

A Redo Hellers procedure is a technically challenging operation especially in these particular patients where surgery was previously done with no details of the previous surgery being available. It is a major operation and demand experience in advance laparoscopic surgery of the esophago-gastric region. Excessive care is required in protecting the vagus, avoiding injury to mucosa and to confirm the completeness of myotomy on table. Endoscopic dilatation may be still being required at intervals in some patients. Hellers with a fundoplication

offers a reduction in symptoms, lesser morbidity, shorter hospital stays and reduction in symptoms post-operatively without the need for a radicle surgery.

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## REFERENCES

1. Schlottmann F, Neto RML, Herbella FAM, Patti MG. Esophageal achalasia: pathophysiology, clinical presentation, and diagnostic evaluation. Am Surg. 2018;84(4):467-72.
2. Abdel Jalil AA, Katzka DA, Castell DO. Approach to the patient with dysphagia. Am J Med. 2015;128(10).
3. O'Neill OM, Johnston BT, Coleman HG. Achalasia: a review of clinical diagnosis, epidemiology, treatment and outcomes. World J Gastroenterol. 2013;19(35):5806-12.
4. Eckardt AJ, Eckardt VF. Current clinical approach to achalasia. World J Gastroenterol. 2009;15(32):3969-75.
5. Akaishi T, Nakano T, Machida T. Clinical usefulness of endoscopy, barium fluoroscopy, and chest computed tomography for the correct diagnosis of achalasia. Intern. Med. 2020;59(3):323-8.
6. Faccani E, Mattioli S, Lugaresi ML, Di Simone MP, Bartalena T, Pilotti V. Improving the surgery for sigmoid achalasia: long-term results of a technical detail. Eur J Cardio-thoracic Surg. 2007;32(6):827-33.
7. Panchanatheeswaran K, Parshad R, Rohila J, Saraya A, Makharia GK, Sharma R. Laparoscopic Heller's cardiomyotomy: a viable treatment option for sigmoid oesophagus. Interact Cardiovasc Thorac Surg. 2013;16(1):49-54.
8. Decker G, Borie F, Bouamrirene D. Gastrointestinal quality of life before and after laparoscopic heller myotomy with partial posterior fundoplication. Ann Surg. 2002;236(6):750-8.
9. Grimes KL, Inoue H, Onimaru M. Double-scope per oral endoscopic myotomy (POEM): a prospective randomized controlled trial. Surg Endosc. 2016;30(5):1344-51.
10. Brunicardi FC, Andersen DK, Billiar TR, Dunn DL, Kao LS, Hunter JG, et al. Schwartz's principles of surgery. Volume 1. 11th Edition. McGraw-Hill Education. 2019;1056.
11. Brunicardi FC, Andersen DK, Billiar TR, Dunn DL, Kao LS, Hunter JG, et al. Schwartz's principles of surgery. Volume 1. 11th Edition. McGraw-Hill Education. 2019;1064.

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