

A Patient with Achalasia with Complication Mega-Esophagus: A Case Report

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ABSTRACT:

A 51-year-old male presented with a history of progressive dysphagia and regurgitation for 6 years. The patient experienced significant weight loss and worsening dysphagia over the past 2 years. A chest X-ray led to a conclusion of esophageal achalasia meanwhile a CT Scan revealed dilation of the cervical, thoracic, and abdominal parts of the esophagus. The patient underwent an initial esophagogastroduodenoscopy (EGD), which revealed a dilated esophageal lumen, a sigmoid-shaped formation in the distal esophagus, and hypertension at the esophago-gastric junction (EGJ). The patient underwent minimal invasive dilation of the EGJ using an endoscopic pneumatic balloon in two sessions one month apart. After a 1-month evaluation post the final EGD, dysphagia symptoms worsened and dilation of an aperistaltic segment from the proximal to distal esophageal lumen was observed. Subsequently, the patient is planned to undergo double set up Heller myotomy surgery and, if necessary, esophageal resection.

Keywords: *Dysphagia, Achalasia, Megaesophagus, Myotomy*

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1. Introduction

Achalasia is a rare oesophageal motility disorder characterized by the failure of the lower oesophageal sphincter (LES) to relax, and absence of peristalsis.¹ Symptoms vary, with the most common including progressive dysphagia, heartburn, regurgitation of undigested food, and respiratory complications, including nocturnal cough and aspiration.¹

Achalasia is a rare condition, with an incidence ranging from 0.63 to 1.63 per 100,000 people.² It is more common in adults than in children, but there is no significant difference between men and women.² Although it is not life-threatening, patients with achalasia often experience a disturbed quality of life because this disease is a lifelong chronic disease.³ If treated properly, patients can improve their quality of life significantly, but if left untreated it can develop into oesophageal lumen dilation, which will later

cause serious complications in the form of megaesophagus.³ About 5% of achalasia patients will experience severe progressive dilation, known as megaesophagus, which often fails to respond to endoscopic pneumatic balloon dilation therapy.⁴

This report describes the case of a patient suffering from achalasia with secondary megaesophagus who underwent endoscopic pneumatic balloon surgery but showed no improvement in dysphagia due to abnormal peristalsis and impaired esophageal emptying, and aims to highlight the importance of early recognition and accurate diagnosis of this condition to allow timely selection of appropriate definitive interventions such as double-set-up Heller myotomy and, if necessary, esophageal resection.

2. Case Report

A 51-year-old male presented with a history of progressive dysphagia and regurgitation for 6 years. The patient experienced significant weight loss and worsening dysphagia over the past 2 years. Laboratory evaluations were within normal limits. A chest X-ray led to a conclusion of

esophageal achalasia, with cardiac and pulmonary evaluations being normal. A contrast-enhanced Thorax CT Scan revealed dilation of the cervical, thoracic, and abdominal esophagus with wall thickening at the gastroesophageal junction, causing luminal narrowing from the cervical to the abdominal esophagus.

On examination, the patient was alert but weak, with stable vital signs. The patient underwent an initial esophagogastroduodenoscopy (EGD), which revealed a dilated esophageal lumen, a sigmoid-shaped formation in the distal esophagus, and hypertension at the esophago-gastric junction (EGJ). The patient underwent minimal invasive dilation of the EGJ using an endoscopic pneumatic balloon in two sessions one month apart. At the final EGD evaluation, no resistance was found at the EGJ and symptoms improved, but after a 1-month evaluation, dysphagia symptoms worsened and dilation of an aperistaltic segment from the proximal to distal esophageal lumen was observed. Subsequently, the patient is planned to undergo double set up Heller myotomy surgery and, if necessary, esophageal resection



Fig 1. Chest X-Ray Examination

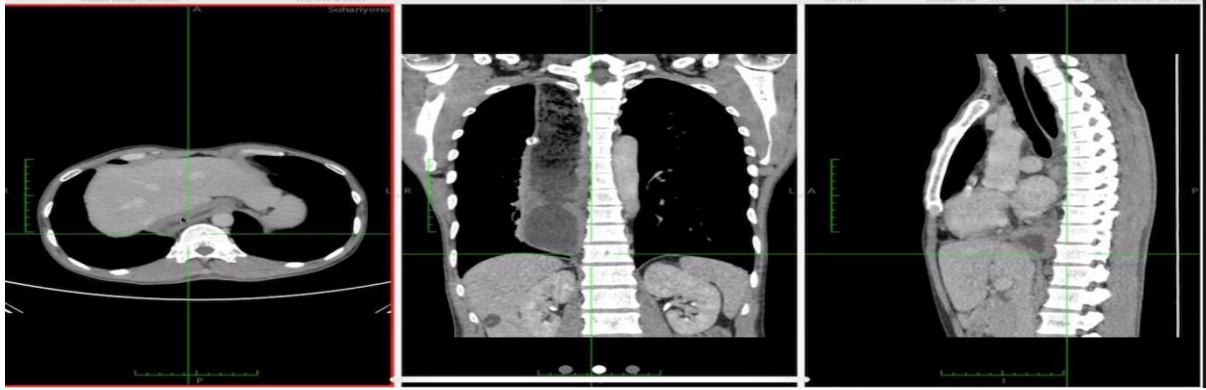


Fig 2. CT Scan Thorax With Contrast

3. Discussion

Achalasia is a rare disease whose cause is not yet fully understood. Several studies have suggested the possibility of an immune disorder leading to chronic ganglionitis.⁵ Myenteric neurons play a role in mediating and coordinating oesophageal peristalsis and lower oesophageal sphincter (LOS) relaxation.⁵ In achalasia, the number of these myenteric neurons is reduced or lost, resulting in aperistalsis and impaired LOS relaxation.⁵ This inflammatory process ultimately results in damage to neurons that release nitric oxide in the myenteric plexus and LOS nerve fibers. This ultimately leads to impairment of cholinergic neurons, which then progresses to the loss of inhibitory nitric oxide neurons, leading to impaired LOS relaxation.³ Other findings suggest a pathologic mechanism for achalasia caused by viruses. Based on available evidence, the most well-known viral infections associated with achalasia are the herpesvirus family (herpes simplex virus, Epstein-Barr virus, Varicella-Zoster virus, and Cytomegalovirus), paramyxovirus, and human immunodeficiency virus (HIV).³ Another study showed an association between achalasia and genetics. Some of the genes identified include HLA class II molecules, vasoactive intestinal peptide receptor 1, KIT, interleukin 10 promoter, and interleukin 23 receptor.⁵ Furthermore, a familial achalasia was found, supporting the possibility of a genetic factor in the pathogenesis of achalasia.⁵ In this case report, the patient had experienced dysphagia for 6 years, that suggests the possibility of chronic ganglionitis that the patient experienced, which then developed achalasia. However, the

exact history of diseases such as viral infections or a family history of achalasia is unknown.

The best initial diagnosis of achalasia is a barium esophagogram or barium swallow.⁶ Typical findings on this exam include a "bird's beak" appearance, with dilation of the proximal oesophagus and the absence of peristalsis on fluoroscopy.⁶ Other recommended tests include an upper endoscopy or esophagogastroduodenoscopy (EGD) to exclude premalignant or malignant oesophageal lesions.⁶ The disadvantage of an EGD is that it can appear normal in the early stages, making it less accurate for early diagnosis.⁶ Findings in advanced cases include a rosette appearance of the esophagogastric junction or an oesophagus that has become dilated, tortuous, and atonic, often with retained food and saliva.⁶ The gold standard for diagnosis of achalasia is oesophageal manometry.⁶ Manometry will reveal incomplete lower oesophageal sphincter relaxation in response to swallowing, sometimes a lack of peristalsis in the lower oesophagus, and an increase in pressure of the lower oesophageal sphincter.⁶

In this present case, the patient performed an EGD examination that revealed a dilated oesophageal lumen, a sigmoid-shaped formation in the distal oesophagus, and hypertension at the esophago-gastric junction (EGJ). This indicated that the stage of achalasia was severe, with complications such as megaesophagus. It is unclear why some patients with achalasia develop megaesophagus; however, a recent study suggests an imbalance between substance P and vasoactive intestinal polypeptide.¹⁰ Symptoms are usually substernal chest pain with dysphagia; however,

syncope can be induced from mechanical irritation of the Vagus nerve.¹⁰ Treatment for megaesophagus is usually esophagectomy and should be performed by an experienced surgeon.¹⁰

Achalasia treatment options are divided into nonsurgical and surgical.⁷ The goal of this therapy is to relieve achalasia symptoms by reducing outflow resistance caused by failure of LOS relaxation.⁷ Nonsurgical therapies include pharmacological interventions, botulinum toxin injections, and pneumatic dilation.⁷ Pharmacologically, medications that can be administered include nitrates, calcium channel blockers, and phosphodiesterase-5 inhibitors.⁷ All three have the effect of reducing LOS pressure and helping to relax the oesophageal muscles.⁷ However, this therapy is temporary and less effective, so it is only given to patients awaiting definitive therapy such as pneumatic dilation or surgery.⁷ Another option is botulinum toxin injection, which works by inhibiting acetylcholine release in the LOS.⁸ This therapy is typically used in high-risk patients awaiting definitive therapy or who have relapsed after myotomy.⁸ This treatment is also short-term, with effectiveness lasting only 6-12 months, and may affect the success of myotomy.⁸

The most effective nonsurgical treatment option for achalasia to date is pneumatic dilation.⁹

This therapy uses an endoscopic procedure to inflate a polyethylene balloon to relax the tight LOS muscle.⁹ Initial dilation in most patients begins with a 3.0 cm balloon, followed by evaluation in 4-6 weeks.⁸ If the patient remains symptomatic, dilation is continued with a larger balloon.⁸ This serial pneumatic dilation approach has demonstrated high success rates.⁸ Several studies have shown that symptom improvement after this procedure reaches 50-93% of patients, however 30% of patients have symptom recurrence within 5 years.⁷ The failure rate with pneumatic dilation is higher with male gender, younger patients (with age less than 40), those with pulmonary complications, or failure of one or more previous pneumatic dilations.⁷

This case describes the case of a patient suffering from achalasia with secondary megaesophagus who underwent endoscopic pneumatic balloon surgery but showed no improvement in dysphagia due to abnormal peristalsis and impaired esophageal emptying, and aims to highlight the importance of early recognition and accurate diagnosis of this condition to allow timely selection of appropriate definitive interventions such as double-set-up Heller myotomy and, if necessary, esophageal resection.

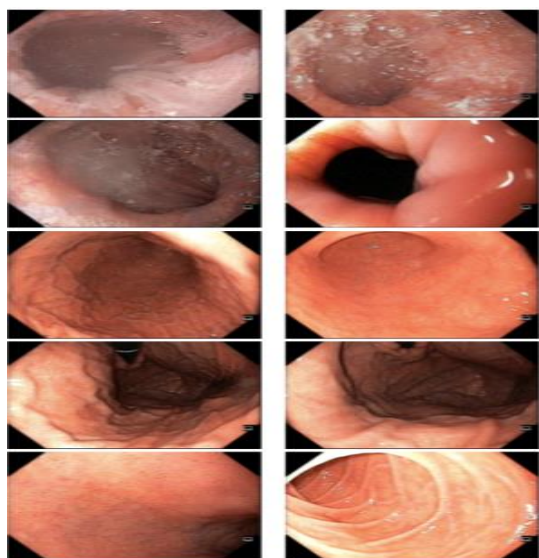


Fig 3. EGJ dilatation with pneumatic balloon endoscope

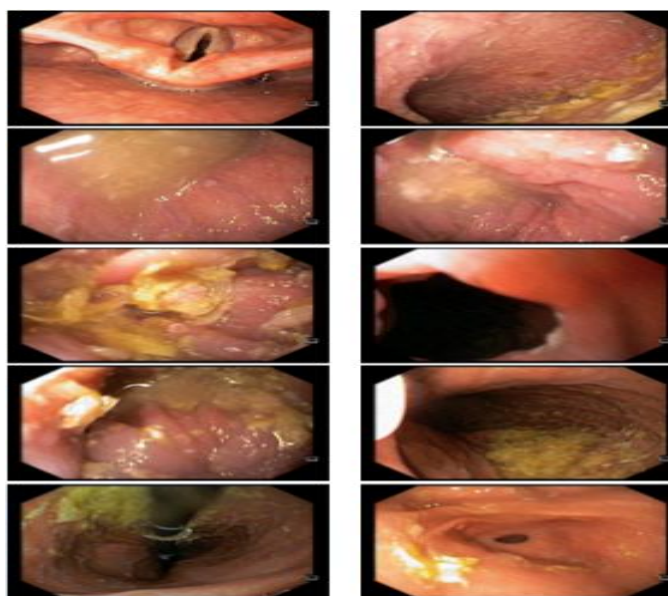


Fig 4. Endoscopy evaluation after dilatation

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