

Robotic Surgery for the Treatment of Achalasia with Intraoperative Manometry: Case Report

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ABSTRACT

Introduction: Achalasia, the Greek term meaning, is a disease of unknown cause in which there is a failure to relax the lower esophageal sphincter and loss of peristalsis in the distal esophagus. Achalasia was first described by Sir Thomas Willis in 1674. **Clinical case:** 63-year-old female with no significant history for the case. Upon admission, the patient presented dysphagia, reflux, aspiration, substernal pain, and weight loss. Endoscopy showed Schatzki ring; Type I hiatal hernia of 5 cm, grade D esophagitis (Los Angeles classification). Manometry showed an IRP -3.06; absent esophageal clearance, without evidence of hiatal hernia, with a

diagnosis of probable achalasia. The SEGD presented an area of V stenosis and a Heller myotomy with Dor-type fundoplication was performed using robotic surgery. Discussion: In clinical practice, the combined use of esophagogastroduodenoscopy (EGD) and upper gastrointestinal series (SGD) helps to evaluate patients who present with dysphagia and/or regurgitation, which are the most common clinical findings in these patients. High-resolution esophageal manometry (HREM) has become an essential tool in the last decade to categorize types of esophageal achalasia with associated lack of relaxation of the lower esophageal sphincter. Current pharmacological, endoscopic and surgical treatment options aim to reduce the hypertonicity of the lower esophageal sphincter, but unfortunately, the absence of esophageal peristalsis does not restore it to normal propulsive waves. Pharmacological treatments, including calcium channel blockers or sequential injection of botulinum toxin, are usually limited to patients who are not amenable to surgical treatment and have limited effects.

Keywords: Achalasia, Heller myotomy, Dor-type fundoplication, Robotic surgery.

INTRODUCTION

Achalasia, the Greek term meaning, is a disease of unknown cause in which there is a failure to relax the lower esophageal sphincter (LES) and loss of peristalsis in the distal esophagus. Achalasia was first described by Sir Thomas Willis in 1674. Treatment of achalasia aims to reduce LES pressure either mechanically by surgical myotomy or endoscopic balloon dilation or pharmacologically by oral nitrites, calcium channel blockers. or endoscopic injection of botulinum toxin. Surgical myotomy, first described by Heller in 1913, has been found to be superior to endoscopic dilation in terms of cumulative response rate with significantly fewer major mucosal tears requiring subsequent intervention [1].

Esophageal achalasia is a dynamic disorder that refers to obstruction of the esophageal outflow tract due to a relaxation of the esophageal sphincter and loss of esophageal peristalsis or spasmodic contraction when the esophageal body or esophagogastric junction is not structurally obstructed. There are two types of achalasia: primary and secondary. Cardiac achalasia is characterized by functional loss of muscle, ganglion cells in the distal esophagus, and LES. The mucosa has long been considered part of the pathogenesis of cardiac achalasia, recent studies have pointed out that inflammation and genetic changes may also contribute to achalasia at the molecular level. Currently, achalasia is a chronic and incurable disease. The different subtypes of achalasia respond differently to drugs and surgical treatments, after which some patients develop submucosal fibrosis; this may relapse and require additional treatment. Achalasia is a relatively rare disease of the esophagus with an incidence of 2.9 per 100,000 adults and 0.11 per 100,000 children and a male-to-female ratio of approximately 1:1. However, recent studies have shown that the incidence of achalasia is increasing, particularly in South America. Achalasia has a bimodal age distribution, with most patients being between 20 and 40 or 60-70 years old. Studies have also shown that the incidence of this disease increases with age [2-5].

CLINICAL CASE

63-year-old female with a history of Diabetes mellitus for 10 years and Systemic Arterial Hypertension for 20 years under treatment. Upon admission, the patient presented dysphagia, reflux, aspiration, substernal pain, and weight loss. Endoscopy showed Schatzki ring; Type I

hiatal hernia of 5 cm, grade D esophagitis (Los Angeles classification). Manometry showed an IRP -3.06 (The IRP is the lowest average pressure at the esophagogastric junction during 4 of the 10 seconds mentioned, which can be continuous or discontinuous in a swallow. An IRP > 15 mmHg means increased resistance to transit of the bolus at the esophagogastric junction and is considered pathological); absent esophageal clearance, without evidence of hiatal hernia, with a diagnosis of probable achalasia (Fig. 1). The SEGJ presented an area of V-shaped stenosis and Heller myotomy with Dor-type fundoplication was performed using robotic surgery (Figs. 2-4).

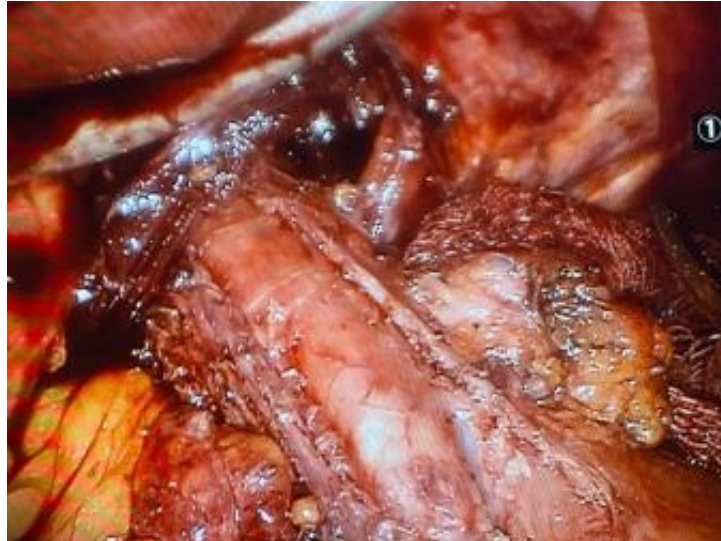
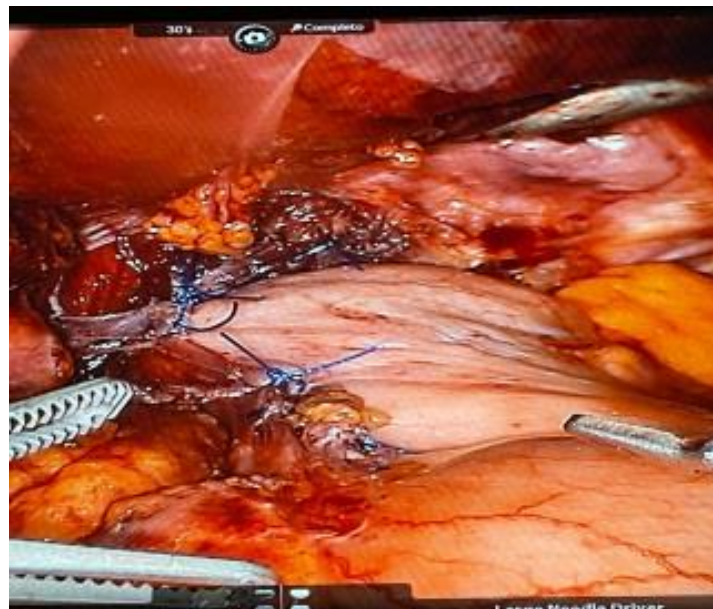


Figure 1: Heller's myotomy.





Figures 2 and 3: Dor's fundoplication

DISCUSSION

Esophageal achalasia represents a primary disorder of esophageal motility secondary to the loss of inhibitory nitrergic neurons at the level of the myenteric plexus. It is characterized by lack of peristalsis and absence or partial relaxation of the LES in response to swallowing during esophageal manometry. Its incidence is estimated at 1 per 100,000 people, while the prevalence is quantified around 10 per 100,000. A cascade of events ranging from possible viral infection or environmental factors lead to inflammation of the esophageal myenteric plexus, leading to autoimmune disease in genetically susceptible individuals and chronic inflammation with destruction of the myenteric plexus. In clinical practice, the combined use of esophagogastroduodenoscopy (EGD) and upper gastrointestinal series (SGD) helps to evaluate the patient who presents with dysphagia and/or regurgitation, which are the most common clinical findings in these patients. High-resolution esophageal manometry (HREM) has become an essential tool in the last decade to categorize types of esophageal achalasia with associated lack of relaxation of the lower esophageal sphincter. HREM helps diagnose three types of motility patterns in esophageal achalasia. Type I is characterized by simultaneous premature contractions that failed to elicit effective peristalsis at all; Type II is classified according to the panesophageal pressurization pattern, and type III is defined as vigorous achalasia due to high-amplitude simultaneous contractions. It is believed that these patterns could be evolving from one type to another, making a different diagnosis of esophageal achalasia at the time of HREM. Current pharmacological, endoscopic and surgical treatment options aim to reduce the hypertonicity of the lower esophageal sphincter, but unfortunately, the absence of esophageal peristalsis does not restore it to normal propulsive waves. Pharmacological treatments, including calcium channel blockers or sequential injection of botulinum toxin, are usually limited to patients who are not amenable to surgical treatment and have limited effects.

Endoscopic pneumatic dilation, Heller myotomy and Dor fundoplication or, more recently, peroral endoscopic myotomy (POEM) are the most commonly used modalities in the treatment of this disorder. Surgery, compared to medical or endoscopic pneumatic dilation, has been shown to provide the best long-term clinical outcome.

While there have been significant improvements in laparoscopic instruments over the years, they are still limited in their ability to perform surgeries in tight spaces or challenging angles, including esophageal myotomy. The recent introduction of robotic surgery reduces these limitations by granting the user greater movement dexterity with the advent of wrist instruments [6].

Key symptoms include dysphagia, regurgitation, chest pain, and weight loss typically quantified by the Eckardt score that presents the frequency and/or intensity of these features. It is a valuable tool to evaluate the clinical outcome after surgical or endoscopic treatment. EGD is commonly performed initially to exclude secondary forms of achalasia due to carcinoma or inflammation of the esophagogastric junction, frequently followed by a barium esophagogram showing the "bird's beak sign" or the typical sigmoid-shaped esophagus in disease in late stage. Therapeutic options include medication. (Calcium channel blockers, nitroglycerin), botulinum toxin injection, pneumatic dilation or peroral endoscopic myotomy. The surgical gold standard has been minimally invasive Heller myotomy with partial fundoplication for many decades, reducing resting pressure in the LES by dividing its longitudinal and circular smooth muscle fibers. Permanent postoperative improvement of dysphagia can be seen in 85 to 100% of patients. With the rise of robotic-assisted surgery over the past two decades due to improved 3D visualization, anti-tremor filters, and greater freedom of movement, similar short-term results have been demonstrated with an even lower rate of intraoperative esophageal perforations [7].

Sectioning muscle fibers of the esophageal sphincter, the so-called Heller myotomy (HM), currently represents the preferred surgical approach for the treatment of achalasia. In particular, laparoscopic MH is today widely accepted as safe and effective. However, recurrence rates of up to 10-25% and intraoperative esophageal mucosal perforation rates ranging between 4 and 20% are reported in the literature. In the last years

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