

## Laparoscopic Heller Cardiomyotomy in the Visceral Surgery Department: About 5 Cases at the Hassan II University Hospital of Fez

Diallo Mamadou<sup>1\*</sup>, Fryse François Mve Okoue<sup>1</sup>, Mohammed El Emin Taleb Maouloud<sup>1</sup>, Kébé Fatoumata Binta<sup>1</sup>, Cissé Mohamed Yaya<sup>1</sup>, Sow Rouguiatou<sup>1</sup>, Diallo Fatoumata Djouldé<sup>1</sup>, Diallo Elhadj Djiba D. H<sup>1</sup>, Abdeslam Bouassria<sup>1</sup>, Hicham El Bouhaddouti<sup>1</sup>, Ouadji Mouaquit<sup>1</sup>, El Bachir Benjelloun<sup>1</sup>, Khalid Ait Taleb<sup>1</sup>, Ousadden Abdelmalek<sup>1</sup>

<sup>1</sup>Department of Visceral Surgery A Hassan II Hospital and University Centre in Fez, Sidi Mohamed Ben Abdallah University, Faculty of Medicine and Pharmacy, Morocco

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\*Corresponding author: Diallo Mamadou

Department of Visceral Surgery A Hassan II Hospital and University Centre in Fez, Sidi Mohamed Ben Abdallah University, Faculty of Medicine and Pharmacy, Morocco

### Abstract

### Case Series

Achalasia is a primitive motor disorder of the esophagus characterized by a defect in relaxation of the lower esophageal sphincter (LES) and a complete absence of esophageal peristalsis. The etiology of this pathology is unknown. Clinical presentation is dominated by dysphagia, often associated with food regurgitation, more rarely with chest pain. Pyrosis may also be present. Symptoms have usually been evolving for many years, progressively worsening, and may be responsible for significant weight loss. Weight loss of more than 10kg, recent onset of symptoms (less than a year), and age over 60 should prompt a search for underlying neoplasia (pseudo-neoplastic achalasia). The diagnosis of achalasia was established on clinical, radiological and endoscopic grounds, in search of associated lesions. All patients underwent a Heller extra-mucosal oesocardiomyotomy associated with an anti-reflux device by cardioplasty. The post-operative course was straightforward in 4 cases, and complicated in 1 case by an iatrogenic breach of the esophageal mucosa, which was subsequently repaired. Functionally, dysphagia, regurgitation and retrosternal pain disappeared, with excellent results. The aim of this retrospective study was to investigate the immediate and remote results of surgical treatment of achalasia using the Heller technique.

**Keywords:** Achalasia, dilatation, esocardiomyotomy, extra-mucosal, manometry, oesogastroduodenal fibroscopy, Heller surgery.

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## INTRODUCTION

Achalasia, or megaesophagus, is a primitive disorder of esophageal motricity affecting patients of both sexes and all races [1]. It is characterized by aperistalsis of the esophageal body with defective relaxation of the lower esophageal sphincter (LES) in response to swallowing. Inflammatory neuropathy, of unknown cause, is probably at the origin of the disease [2]. Since its first description in 1672 by Thomas Willis [1], this condition has been the subject of several studies concerning its pathophysiology; the most recent are based on manometry data [2]. Typical symptoms include dysphagia, regurgitation and retrosternal pain. Esophageal manometry remains the gold standard for diagnosis [2]. In achalasia, the therapeutic objective is to reduce or eliminate the functional obstacle in the lower esophagus. This can be achieved by several therapeutic modalities: Heller-type extra-mucosal longitudinal oesocardiomyotomy, by conventional surgery [3], by

laparoscopic or thoracoscopic surgery [4], by per-endoscopic progressive pneumatic dilatation [5], and recently by endoscopic intrasphincteric injection of botulinum toxin [6]. HELLER cardiomyotomy (HCM) can be performed openly or laparoscopically. Studies comparing these two approaches have shown that there are no differences in terms of outcomes and postoperative complications [7]. Nevertheless, laparoscopy had multiple advantages: shorter hospital stay, less blood loss, lower consumption of analgesics postoperatively, better aesthetic result and early return to professional activities offsetting its higher initial cost. This has led many authors to consider laparoscopic HCM as the gold standard treatment for patients suffering from achalasia [8].

However, the use of this approach remains marginal in our context. We report 5 clinical cases operated by extra-mucosal longitudinal myotomy

according to Heller, and our experience in the treatment of achalasia by laparoscopy.

## PATIENTS AND OBSERVATION

### Case 1

Patient aged 38, with no significant pathological history. Admitted with dysphagia to semi-liquids and solids

Onset of symptomatology dates back to 2019 with the onset of food clinging, never explored, which progressively worsened, in 2021 with the onset of dysphagia to solids, semi-liquids and solids, motivating the patient to consult a private practice where a standard biological workup and an upper GI endoscopy were performed. The patient underwent medical treatment with no favourable outcome, and was then referred to us for surgical management.

According to the anamnestic elements, the clinical examination revealed a calm patient in good general condition, with algic vomiting. She was eupneic, hemodynamically and respiratorily stable, with blood pressure 110/70mmHg, heart rate 93/min, pulse ox 98% and temperature 37°C.

Physical examination revealed a soft, excavated abdomen with epigastric tenderness. The rest of the somatic examination was unremarkable.

The standard laboratory workup revealed a hydroelectrolyte disturbance, with kalemia at 2.8 and a correct Natremia. No biological inflammatory syndrome.

An oeso-gastroduodenal fibroscopy with biopsy was performed, showing a tight stenosis of the cardia. Anatomopathology: non-specific, no evidence of malignancy. Esophageal manometry: Chicago type achalasia

Given that the patient had undergone medical and endoscopic treatment by a gastroenterologist with no favourable outcome, the decision was made to carry out surgical treatment using a laparoscopic approach.

Surgical treatment consisted in performing an extra-mucosal Heller myotomy with an anterior hemi valve. Postoperative follow-up was straightforward.

### Case 2

Patient aged 59, followed for ischemic heart disease on sintrom and Avlocardil for several years. Admitted for surgical management of esophageal motor disorder, evolving for one year.

Symptomatology began a year ago with the onset of dysphagia to liquids, semi-solids and progressively to solids, in a context of weight loss for which she had received medical and endoscopic

treatment without favorable outcome, then transferred to us for further management in our facility.

The patient arrived conscious, with signs of severe dehydration. Hemodynamically and respiratorily stable, with blood pressure 109/80 mm Hg, heart rate 90/min, respiratory rate 20 cycles/min.

Abdomen tender in epigastric region. An oesogastroduodenal fibroscopy was performed, showing cardiac stenosis plus megaesophagus.

Esophageal manometry showed: relaxation of the LES and esophageal peristalsis, suggesting esophageal achalasia.

Standard laboratory tests: not essential for diagnosis, but useful for the patient's overall assessment and preoperative work-up.

Complete blood count, platelets, iron-deficiency anemia with hemoglobin at 10g/dl, absence of biological inflammatory syndrome. Hydroelectrolytic disorder with hypokalemia at 2 mmol/l and hyponatremia at 130 mmol/l. Correct haemostasis.

The patient's management required her to be taken to the intensive care unit for conditioning for two days prior to surgery.

Surgical treatment consisted of Heller seromyotomy and DOOR fundiolicature via laparoscopic approach.

On the third day of surgery, the patient underwent upper gastrointestinal opacification as part of surveillance, which revealed no abnormalities. Discharged on the 6th day of hospitalization. Postoperative follow-up was straightforward.

### Case 3

Patient aged 27, with no notable pathological history. Followed up for pyrosis and regurgitation by a gastroenterologist in private practice under medical treatment, with no favorable outcome and the appearance of dysphagia with liquids, semi-solids, then solids, evolving for 3 years. Referred to us for further treatment

On initial examination, the patient was conscious, in good general condition, vomiting, eupneic and hemodynamically and respiratorily stable, with blood pressure at 11/7 mmHg, heart rate at 90/min, room air saturation at 98% and temperature at 36°C.

Physical examination revealed a soft, excavated abdomen; tenderness in the epigastric region. Oeso-gastroduodenal fibroscopy: antrofundial gastritis. Oesogastroduodenal fibroscopy and transit: megaesophagus.

Oesophageal manometry: disturbed relaxation of lower oesophageal sphincter and oesophageal peristalsis. Appearance in favour of achalasia.

A standard biology work-up is not useful for diagnosis, but it is useful for the overall work-up, as well as for the preoperative work-up.

Blood count, platelets: hemoglobin 13g/dl, no biological inflammatory syndrome. Blood ionogram: hypokalemia 2.8 mmol/l; hyponatremia 130 mmol/l. Renal function correct, with uremia at 0.10 g/l,

Creatinemia 8 g/l. Correct haemostasis. The patient was conditioned by anaesthetists to correct hydroelectrolytic disorders for two (02) days. Surgical management consisted of: Laparoscopic approach

Exploration for the absence of intra-abdominal effusion; Heller extra-mucosal myotomy with an anterior hemi valve. Postoperative follow-up was straightforward, with discharge authorized on the 6th postoperative day. Reviewed at postoperative check-up with no complaints.

#### Case 4

Patient aged 34, followed for megaesophagus in OUJDA, having benefited from several dilatation sessions without improvement of his clinical symptomatology, who is transferred to the CHU of Fès for management of dysphagia to liquids, semi-solids and then solids evolving for several years according to the patient's entourage.

According to the anamnestic elements, the examination revealed a hemodynamically and respiratorily stable conscious patient with a blood pressure of 13/8mmHg, a heart rate of 96 per minute, a respiratory rate of 20 cycles per minute and a temperature of 37°C.

Physical examination cachectic patient with an excavated abdomen tender in the epigastric region. Oesogastroduodenal transit: Appearance in favour of lower oesophageal stenosis with upstream dilatation suggestive of achalasia.

Esophageal manometry: Lower esophageal sphincter relaxation disorder suggestive of esophageal achalasia. Standard laboratory tests: not useful for diagnosis, but useful for the patient's overall assessment and preoperative work-up.

Blood count, platelets: iron-deficiency anemia at 10g/dl; absence of biological inflammatory syndrome. Complete blood ionogram: hydroelectrolytic disorder with hypokalemia at 2.5 mmol; hyponatremia at 130 mmol.

Patient requires conditioning and correction of fluid and electrolyte disorders. Surgical management consists of: laparoscopic approach

Section of the left triangular ligament of the liver and of the longitudinal and circular muscle fibers of the cardio-oesophageal junction.

Extra-mucosal Heller myotomy with an anterior hemi-valve. Postoperative follow-up was straightforward. A gastrographine transit did not reveal a fistula, and a liquid diet was authorized.

On the twelfth postoperative day, the patient presented with abdominal bloating, fever and subcutaneous emphysema. He was resected by median laparotomy: abundant lavage plus suture of the 2 cm-long breach of the lower esophagus.

Intraoperative oesogastroduodenal water-soluble opacification: no fistulous tract or extravasation of contrast medium. Moderate esophageal dilatation without stenosis.

#### Case 5

Patient aged 40, with no pathology of note. Admitted for management of pyrosis, regurgitation plus fluid dysphagia, which had been present for 5 years.

Symptoms began 5 years prior to admission with the onset of regurgitation plus pyrosis occurring postprandially, aggravated by bending forward without other associated disorders. He consulted a gastroenterologist where he received symptomatic treatment, with no favorable outcome, and the worsening of these symptoms, dysphagia to liquids, semi-solids and solids.

According to the anamnestic elements, the clinical examination found a conscious patient, hemodynamically and respiratorily stable, with normo colored integuments and conjunctiva, blood pressure 13/8 mm hg, heart rate 100 per minute, respiratory rate 18 cycles per minute, temperature 37°C.

Physical examination revealed a supple abdomen, with tenderness in the epigastric region. The rest of the somatic examination was unremarkable.

Oesogastroduodenal fibroscopy: 4 cm hiatal hernia, erythematous antero-fundal gastritis. Biopsy: result in favour of chronic active atrophic antral gastritis + absence of *Helicobacter pylori*.

Esophageal manometry: moderate hypotonia of lower esophageal sphincter. PH metry: normal Oesogastroduodenal transit: sliding hiatal hernia with gastro-oesophageal reflux.

Standard laboratory tests: not useful for diagnosis, but necessary for the patient's overall assessment and preoperative work-up.

Blood count and platelets: hemoglobin level 14g/dl, absence of biological inflammatory syndrome. Correct renal function, complete ionogram with correct kalemia at 3.5 mmol, natremia at 140, normal hepatic workup.

Surgical treatment consisted of: Laparoscopic approach. Heller extra-mucosal myotomy plus NISSEN fundoplication.

## DISCUSSION

Esophageal motor disorder defined by the absence of propagated contractions (aperistalsis) at the level of the body of the esophagus and by a defect in relaxation of the lower esophageal sphincter (LES) in response to swallowing (achalasia). Typical symptoms include dysphagia, regurgitation and retrosternal pain [9].

The spontaneous evolution of the disease leads to progressive dilatation of the esophagus (megaesophagus).

It is a rare condition: its incidence has been estimated at around 0.5/100,000 inhabitants per year, with a prevalence of 8/100,000 [10]. Achalasia affects both sexes and all ages, but is rare in children. Epidemiological studies highlight the role of geographical, environmental and socio-economic factors in the development of the disease [11].

Of the symptoms encountered, dysphagia is constant. It is the most common and earliest symptom of the disease [12, 8], as is the case in our series. Our 5 patients presented with dysphagia. In the initial period of the disease, it is variable and capricious, aggravated by stress. This dysphagia is sometimes paradoxical, i.e. elective for liquids, whereas solids pass through well. Chest pain is mainly observed in the initial period of the disease [14]; in our study, all patients had this symptom. These pains are related to the anarchic contractions of the achalasic oesophagus and are sometimes triggered by the ingestion of iced beverages [3]. These pains are constant in vigorous forms of achalasia [9, 10]. Regurgitation is the second most frequent symptom of this condition [8].

In our series, all patients presented with these symptoms. Regurgitation may be active, resulting from post-prandial contractions of the achalasia esophagus. They can also be passive, caused by anteflexion and decubitus. Finally, weight loss is frequent, sometimes considerable [3, 13], as a result of inadequate nutrition. In our series, weight loss was insignificant. Fibroscopy may show a protrusion of the cardia, but is not sufficient to make the diagnosis. Esophageal manometry shows a lack of relaxation of the esogastric junction and an

absence of esophageal peristalsis during swallowing, confirming the diagnosis. Treatment of achalasia consists in removing the obstruction at the lower esophageal sphincter. Pharmacological treatments (nitrates and calcium channel blockers) are not very effective. Surgical treatment aims to sever the muscle fibers of the lower esophageal sphincter, permanently correcting the hypertonia.

Laparoscopic anterior extra-mucosal cardiomyotomy, or Heller myotomy, was described in 1991, and has become a reference for its efficacy [1, 2]. The length of the myotomy is 8 to 10cm, 6cm over the esophagus and 2cm over the stomach. The circular and longitudinal muscle layers are cut until the mucosa is identified. Acid reflux often complicates the procedure, which is prevented by performing a fundoplication.

Technically, Heller's extra-mucosal longitudinal oesocardiomyotomy was the treatment of choice for all our patients. It consists of an anterior myotomy, which transects all the muscle fibers of both layers of the lower esophagus and cardia. In this respect, the myotomy must be sufficiently long upwards, for around 8 cm from the oesogastric junction, and extend for 2 to 3 cm below on the anterior surface of the greater tuberosity in contact with the deep muscle fibres, which are not only circular but also oblique (Helvetius tie) [15].

Postoperative follow-up was straightforward in 4 cases. In one case, the post-operative course was complicated by an iatrogenic breach of the esophageal mucosa, unrecognized intraoperatively. Esophageal perforation is the main intraoperative complication when performing an extra-mucosal Heller cardiomyotomy. It occurs in 7% to 10% of cases during the laparoscopic approach [16].

This breach required a repeat operation and was treated by laparotomy, a simple suture plus peritoneal toilet.

Functionally, we achieved disappearance of dysphagia, regurgitation, retrosternal pain and weight regain in all patients.

Postoperative esophageal barium transit showed in all cases regression of esophageal dilatation and good barium passage.

The evolution was favorable in all cases and complicated in 1 case by iatrogenic breach of the esophageal mucosa, repaired secondarily. Hospital stay ranged from 7 to 8 days.

## CONCLUSION

To this day, Heller cardiomyotomy remains the reference treatment for achalasia. Given its many advantages, laparoscopy is the recommended approach for this myotomy. Its use should therefore be encouraged

in our context. An anti-reflux procedure should always be added.

**Conflict of Interest:** The authors declare no conflicts of interest.

**Authors' Contributions:** All authors have contributed to the development of the work and endorse the document.

They have also read and approved the final version of the manuscript.

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