

**A COMPARATIVE STUDY OF  
MEDICAL VERSUS LAPAROSCOPIC  
MANAGEMENT OF  
GASTROESOPHAGEAL REFLUX DISEASE**

---

THESIS

SUBMITTED IN FULFILMENT  
FOR THE MASTER DEGREE IN  
GENERAL SURGERY

BY

MOHAMED IBRAHIEM EL-ETRIBY  
M.B.B.C.H

SUPERVISORS

**Dr. ANTOINE HALEPIAN**  
PROFESSOR OF GENERAL SURGERY  
FACULTY OF MEDICINE  
CAIRO UNIVERSITY

**Dr. HESHAM MAHMOUD AMER**  
ASSISSTANT PROFESSOR OF GENERAL SURGERY  
FACULTY OF MEDICINE  
CAIRO UNIVERSITY

(2007)

## **Acknowledgment**

I would like to express my deep appreciation to my supervisors for their meticulous professional assistance and encouragement.

I'm grateful to Professor Dr. Antoine Halepian, professor of general surgery, faculty of medicine, Cairo University for his constant effort, kind guidance, close supervision and support.

I'm deeply indebted to Professor Dr. Hesham Amer, assistant professor of general surgery, faculty of medicine, Cairo University for his valuable advice, close supervision and generous efforts throughout this work.

Lastly thanks to everyone who supported me in this work.

## **Abstract**

The study involves prospective and retrospective GERD of 40 patients with symptomatic GERD grades (II, III , IV) who will be allocated to one of two groups:

Group A: 20 patients undergoing medical treatment

Group B: 20 patients undergoing laparoscopic treatment of their acid reflux

Pre-study will be compared with post-study with a 9 Month follow up, depending on:

- Symptomatic relief
- Healing of oesophagitis
- Recurrence (judged by endoscopy)
- Occurrence of complications
- Tolerance to medical treatment

Comparison between groups would be carried using chi-squared analysis for non numerical data.

In this study, there's obvious significant improvement in the lower esophageal sphincter (LES) length and LES pressure in the surgical group, with a concomitant improvement in healing of esophagitis and symptomatic relief.

Concerning the LES length and LES pressure a significant increase in both length and pressure where noted in the post surgical group.

Concerning the healing of esophagitis, surgery proved to be superior to medical treatment in healing of esophagitis, while surgery succeeded in stepping down the stage of esphagitis to 80% having no

esophagitis at all & 10% in stage I & another 10% in stage II, medication left 70% in stage II & 20% is stage III.

Concerning symptomatic relief, surgery also proved to be superior to medical therapy in relieving symptoms of GERD, especially regurgitation, with a success of 100% in the surgery group, while medication failed to relief regurgitation in any patient who suffered this symptom. i.e.(0% success).

Surgery was found to be slightly superior to medication in relieving heart burn with a success percentage of 80% compared to 60% in the medical group, and a lesser significance in reliving dysphagia in which success percentage was 80% in the surgical group compared to 66.6% in the medical group.

**Key words:**

GERD (gastroesophageal reflux Disease), B.E (Barrett's esophagus), H.H (Hiatus Hernia), PPI (proton Pump Inhibitor), LES (Lower esophageal sphincter), SCJ (Squamocolumar junction), TLESR (Transient lower esophageal sphincter relaxation).

# **Contents**

• Introduction	P.I
• Review of literature:	
• Anatomy.	P.1
• Physiological background to antireflux mechanisms	P.12
• Pathogenesis of GERD.	P.21
• Clinical Picture.	P.31
• Complications.	P.44
• Investigations.	P.50
• Treatment:	
• General measures.	P.72
• Medical.	P.77
• Endoscopic therapy	P.86
• Surgical.	P.91
• Aim of work	P.120
• Patients and methods.	P.121
• Results.	P.126
• Discussion.	P.132
• Conclusion	P.139
• Figures.	P.141
• References.	P.155

# Introduction

## **Introduction**

Gastro-esophageal reflux disease (GERD) is a common chronic disorder that has severe impact on quality of life. Moreover, GERD may also cause different grades of esophagitis and sometimes severe complications, such as ulceration, strictures, Barrett's mucosa, and adenocarcinoma of the esophagus.<sup>1</sup> Surveys revealed that up to 15–20% of adults experience heartburn on a weekly basis and therefore the cost of drugs prescribed for the treatment of GERD represents a heavy economic burden for society.<sup>2</sup> Although proton pump inhibitors (PPIs) are extremely effective in healing esophagitis and improving typical reflux symptoms, they also have shortcomings and limitations.<sup>3</sup> Firstly, they do not restore the normal antireflux barrier at the gastro-esophageal junction and there is frequently a rebound acid hypersecretion after cessation of drug intake which both contribute to the high relapse rate observed after discontinuation of PPI therapy.<sup>4</sup> Finally, a challenging problem remains the treatment of the approximately 10–20% of patients with proven GERD who have only a partial or no response to high doses of PPIs.<sup>5</sup>

Spechler *et al* reported that 62% of patients who underwent antireflux surgery were still taking acid suppressive drug after 10 years.<sup>6</sup> Moreover substantial morbidity and some mortality exist. Complications such as dysphagia, inability to belch, diarrhea, and flatulence may develop in up to 30% of patients.<sup>7</sup>

Laparoscopic fundoplication is often proposed as an alternative and more definitive option, especially in young patients, because it is intended to cure the disorder and the laparoscopic approach makes surgery more acceptable.<sup>8,9</sup>



## References

1. **Lagergren J** , Bergström R, Lindgren A, *et al.* Symptomatic gastroesophageal reflux as a risk factor for esophageal adenocarcinoma. *N Engl J Med* 1999;**340**:825–31.
2. **Locke GR III**, Talley NG, Fett SL, *et al.* Prevalence and clinical spectrum of gastroesophageal reflux: a population-bases study in Olmsted County. *Gastroenterology* 1997;**112**:1448–56.
3. **Tytgat GN**. Shortcomings of the first-generation proton pump inhibitors. *Eur J Gastroenterol Hepatol* 2001;**13** (suppl 1) :S29–33.
4. **Bardhan KD**. The role of the proton pump inhibitors in the treatment of gastroesophageal reflux disease. *Aliment Pharmacol Ther* 1999;**9** (suppl 1) :15–25.
5. **Pegini PL**, Katz PO, Castell DO. Ranitidine controls nocturnal gastric acid breakthrough on omeprazole: a controlled study in normal subjects. *Gastroenterology* 1998;**155**:1335–9.
6. **Leite L** , Johnston B, Just R, *et al.* Persistent acid secretion during omeprazole therapy: a study of gastric acid profiles in patients demonstrating failure of omeprazole therapy. *Am J Gastroenterol* 1996;**91**:1527–31.
7. **Lundell L** . Surgical treatment of gastroesophageal reflux disease. In: Orlando, ed. *Gastroesophageal reflux disease*. New York: Marcel Dekker Inc, 2000:311–31.
8. **Lundell L** . Laparoscopic fundoplication is the treatment of choice for gastro-esophageal reflux disease. *Gut* 2002;**541**:468–71.
9. **Galmiche JP**, Zerbib F. Laparoscopic fundoplication is the treatment of choice for gastro-esophageal reflux disease. *Gut* 2002;**541**:472–4. .

## **Abbreviations used in this work**

<b>B.E</b>	Barrette's esophagus
<b>cAMP</b>	Cyclic adenosine mono phosphate
<b>CLEO</b>	Columnar epithelium lined esophagus
<b>DES</b>	Distal esophageal stenosis
<b>EGF</b>	Epidermal growth factor
<b>EGJ</b>	Esophago-gastric junction
<b>ELGP</b>	Endo luminal gasroplcation
<b>EUS</b>	Endoscopic ultrasound
<b>GERD</b>	Gastro esophageal reflux disease
<b>H.H</b>	Hiatus hernia
<b>H.pylori</b>	Helicobacter pylori
<b>H<sub>2</sub>RA</b>	H <sub>2</sub> receptor antagonist
<b>HPZ</b>	High pressure zone
<b>LA</b>	Los Angeles
<b>LES</b>	Lower esophageal sphincter
<b>LGD</b>	Low grade dysplasia
<b>NUD</b>	Non-ulcerative disease
<b>OCT</b>	Optical coherence tomography
<b>PPI</b>	Proton pump inhibitor
<b>RFe</b>	Radiofrequency energy
<b>SART</b>	Standard acid reflux test
<b>SCJ</b>	Squamo-columnar junction
<b>TDD</b>	Total duodenal diversion
<b>TLESRs</b>	Transient lower esophageal sphincter relaxations
<b>UES</b>	Upper esophageal sphincter

# Anatomy

# **ANATOMY OF ESOPHAGUS**

## **Embryology**

The esophagus starts to develop on the twentieth day after fertilization as a short tube extending from the tracheal groove to the dilatation of the foregut designed to become the stomach. This tube elongates with the ascent of the larynx and the descent of the heart, during which process, the esophageal lumen becomes temporarily obliterated by a proliferation of the endodermal columnar lining cells. Failure to recanalize is the cause of esophageal atresia. After recanalization, the epithelial lining of the esophagus changes to a stratified squamous type. The muscular and connective tissue coats of the esophagus are derived from the visceral mesoderm between the sixth and twelfth weeks of life. Initially, the vagal trunks run along the side of the esophagus, but as the stomach rotates, the right vagus assumes a position posterior to the cardio-esophageal junction and the left trunk comes to lie anterior to the esophagus (*Peter, 1992*).

## **Adult Anatomy**

The esophagus is a hollow muscular tube guarded by upper and lower sphincters and extends from the lower border of the cricoid cartilage (sixth cervical vertebra) to the stomach. Its length is 25-30 cm although its measurements vary with the built and height of the individual. When

viewed endoscopically, there is a number of normal constrictions, the positions of which are measured by convention from the upper incisor teeth. Thus, the beginning of the esophagus (just distal to the cricopharyngeus) is found at 15 cm from the incisor teeth, the indentations by the aortic arch and left main bronchus occur at 22 and 27 cm respectively, and the cardio-esophageal junction is encountered at 40 cm in the male and 37 cm in the female.

The esophagus descends in front of the lower cervical and thoracic vertebrae but deviates to the left in the neck and then to the right of the midline in the thorax, except at the lower end when it again inclines to the left before passing through the diaphragmatic hiatus in front of the aorta, Fig(1). These deviations from the midline are important surgically in that the cervical esophagus is best approached from the left side and the thoracic portion through a right thoracotomy, except the lower end which is more accessible through a left thoracotomy or thoraco-abdominal approach (*Sleinseger and Fordtran, 1989*).

Descriptively the lower esophagus is divided into: the supradiaphragmatic portion, the inferior esophageal constriction, the vestibule and the cardia.

1- Radiologically, the supradiaphragmatic portion consists of an ampulla and the empty segment just distal. The ampulla is not an anatomical dilatation and is caused by the primary peristaltic wave (during a barium swallow), acting in conjunction with the negative intrathoracic pressure which momentarily expand this segment just before the lower

esophageal sphincter (LES) relaxes.

2- The inferior esophageal constriction consists of a concentric narrowing of the esophageal lumen present at the level of the diaphragmatic hiatus. On average, it is situated some 2 cm from the cardio-esophageal junction. It is not synonymous with the LES although this extends to include this region. The longitudinal esophageal mucosal folds are very prominent inside the inferior constriction but disappear readily when the esophagus is dilated during endoscopy.

3- The intra-abdominal segment of the esophagus is also known as the vestibule. It is often described as an inverted funnel or cone which inclines to the left before joining the stomach at an angle (cardiac angle or angle of Hiss). When viewed endoscopically from within the stomach, it forms a well-marked ridge at the left margin of the gastro-esophageal junction which is referred to as incisura.

4- The cardia denotes the junction between the esophagus and the stomach. The only reliable and constant anatomical landmark of this is made by the sling or oblique fibers of the stomach but these cannot be identified endoscopically. In the clinical context, therefore, the term cardia is used to describe the junctional zone between the esophagus and stomach. It contains the squamocolumnar junction which forms a serrated line (Z-line), marking the abrupt change from the tough, smooth and pale squamous epithelium of the esophagus with the epithelium of the stomach. The Z-line is situated within 1-4 cm of the anatomical gastro-esophageal junction. A zone of junctional epithelium is interposed between the squamous lining of the esophagus and the gastric mucosa of the rest of the

stomach. It is lined by columnar cells, contains simple tubular mucous glands which are superficial to the muscularis mucosa and is resistant to acid and peptic digestion. This junctional epithelium extends upwards in patients with longstanding reflux esophagitis when it is referred to as Barrett's epithelium.

## **Esophageal Attachments**

The esophagus is loosely bound to adjacent structures by fibroareolar tissue throughout its course except at the upper and lower ends where fixation is more secure. Superiorly, the longitudinal muscle fibers of the esophagus are inserted into the cricoid cartilage. The lower attachments consist of serous reflections and the phreno-esophageal membrane. The supradiaphragmatic pleural reflection is continuous with the mediastinal pleura and is separated from the lower segment of the esophagus by a condensation of the endothoracic fascia which constitutes the phreno-esophageal membrane. This important fibro-elastic membrane fixes the lower gullet but permits its continuous vertical displacement which occurs with respiration. The phreno-esophageal membrane consists of a superior and an inferior limb. The latter is inserted into the cardia, and the superior limb into the lower 3.0 cm of the thoracic esophagus. The fibers of the membrane are disposed in bundles and lamellae and are inserted deeply into the esophageal walls, some reaching the submucous layer. As a high percentage (40-60%) of the fibers is made of elastin, the membrane has both strength and resilience which are necessary to cope with the