Nissen's vs Toupet fundoplication in management of GERD

A Thesis

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Presented By

Abdallah Mohamed Saad Orabi

M.B.B.Ch.
Ain-Shams University

Supervised By

Prof. Dr. Amr Abdel-Raouf Abd-Elnasser
Professor of General surgery
Faculty of Medicine - Ain-Shams University

Prof. Dr. Abd-Elrahman Mohamed El-Ghandour
Lecturer of General Surgery
Faculty of Medicine - Ain-Shams University

Faculty of Medicine
Ain-Shams University
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Abdallah Mohamed Saad Orabi

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List of Abbreviations

BMI	:	Body mass index
EE	:	Erosive esophagitis
EMR	:	Endoscopic mucosal resection
ERD	:	Erosive reflux disease
ESD	:	Endoscopic submucosal dissection
GERD	:	Gastroesophageal reflux disease
H2RA	:	H2 receptor antagonist
HRM	:	High-Resolution Manometry
IPG	:	Implantable pulse generator
LARS	:	Laparoscopic Anti Reflux Surgeries
LES	:	Lower esophageal sphincter
LESP	:	Lower esophageal sphincter pressure
MII	:	Multichannel Intraluminal Impedance
NERD	:	Non-erosive reflux disease
PEH	:	Paraesophageal hernia
PPI	:	Proton pump inhibitor
PPIs	:	Proton pump inhibitors
RF	:	Radio-frequency
SCJ	:	Squamocolumnar junction
TLESRs	:	Transient lower esophageal sphincter relaxations

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Introduction

Gastroesophageal reflux disease (GERD), as generally defined, is a common clinical condition that results from the reflux of gastric material through the lower esophageal sphincter (LES) into the esophagus or oropharynx, causing symptoms and/or injury to esophageal tissue that are severe enough to disrupt a patient's life ***(Aziz et al., 2016).***

GERD is much more prevalent in Western countries, represented in Europe and USA than in Asiatic countries. It has been shown that GERD prevalence increases in parallel with the remarkable growth of obesity. It is very frequent in the community and ranges from 10% to more than 30%, according to the various population-based studies ***(Savarino et al., 2017).***

The term encompasses both symptoms and pathophysiologic changes to the esophageal mucosa, which occur as a result of exposure of the distal esophagus to acidic gastric contents after episodes of gastroesophageal reflux ***(Herregods et al., 2015).***

There are two main phenotypic manifestations, erosive reflux disease (ERD) and non-erosive reflux disease (NERD) and the latter includes the majority of patients (up to 70%). the progression from NERD to ERD, from mild to severe ERD and from ERD to Barrett's esophagus may occur only in a small number of cases,

ranging from 0% to 30%, 10-22% and 1-13%, respectively ***(Savarino et al., 2017).***

GERD can manifest in a wide range of symptoms which tend to be more common after meals and are often aggravated by recumbency. Symptoms can be subdivided into typical (heartburn and acid regurgitation), atypical (epigastric pain, dyspepsia, nausea, bloating, and belching) and extraesophageal(chronic cough, asthma, laryngitis and dental erosions) ***(Francis & Vaezi,. 2015).***

Pathophysiology of GERD is multifactorial. Pathologic reflux is thought to occur when the injurious properties of refluxed gastric acid, bile, pepsin, and duodenal contents overwhelm normal esophageal protective antireflux barriers, such as esophageal acid clearance and mucosal resistance. The primary underlying mechanism causing pathologic reflux appears to be a defective LES, which increases the volume of acidic gastric contents that refluxes into the esophagus ***(Herregods et al., 2015)***

While GERD is usually nonprogressive, in a minority of cases disease progression is associated with the development of complications that range from esophagitis, bleeding, esophageal erosions and ulcerations, stricture formation, Barrett's esophagus to adenocarcinoma of the esophagus ***(Dunbar et al., 2016).***

GERD is typically diagnosed by a combination of clinical symptoms, response to acid suppression medication, as well as objective testing with (upper

endoscopy, esophageal pH monitoring, Barium esophagram and esophageal manometry) ***(Katz et al., 2013).***

GERD is a chronic condition requiring continued management using medications and lifestyle modifications. Pharmacotherapy, particularly the use of antisecretory agents, has probably modified the natural history of GERD. Proton pump inhibitor (PPI) use, in particular, has had an enormous impact on treatment, in providing significantly improved erosive esophagitis healing rates and better symptom control. On the other hand, selected patients with severe disease may benefit from surgery to prevent relapse ***(Gyawali & Fass, 2018).***

Surgical therapy is aimed at correcting the anatomic and functional abnormalities of the esophagogastric junction that result in gastroesophageal reflux. Corrective measures include reduction of hiatal hernia, if present, construction of a fundal wrap to augment the lower esophageal sphincter (LES) and increase its resting pressure, and approximation of the diaphragmatic crura ***(Duke &Farrell, 2017).***

Nissen's fundoplication (posterior 360 degree wrap) is the golden standard for surgical treatment of gastroesophageal reflux disease (GERD). This operation involves repairing the hiatal hernia, which is often present, followed by fashioning the fundus of the stomach around the lower esophagus to form an external buttress. However, it is associated with a high incidence of postoperative

complications (dysphagia and gas-bloat syndrome) ***(Su et al., 2016).***

On the other hand Toupet fundoplication (posterior 270 degree wrap) offers equivalent symptom relief and has a significantly lower risk of postoperative complications compared with Nissen's fundoplication ***(Su et al., 2016).***

Aim of the Work

To compare post-operative outcome on quality of life, anatomical failure, symptom relieve & recurrence rate between Nissen's & Toupet fundoplication.

Surgical anatomy of esophagus and diaphragm

Esophagus

Is a muscular tube-like organ, 25–28 cm Long, approximately 2 cm in diameter, located between lower border of laryngeal part of pharynx and cardia of stomach.

Topographically Start and end points of esophagus correspond to 6th cervical vertebra and 11th thoracic vertebra, and the gastroesophageal junction corresponds to xiphoid process of sternum ***(Oezcelik., 2011).***

Esophagus is anatomically divided into three parts: cervical esophagus, thoracic esophagus and abdominal esophagus. **Fig. (1)**

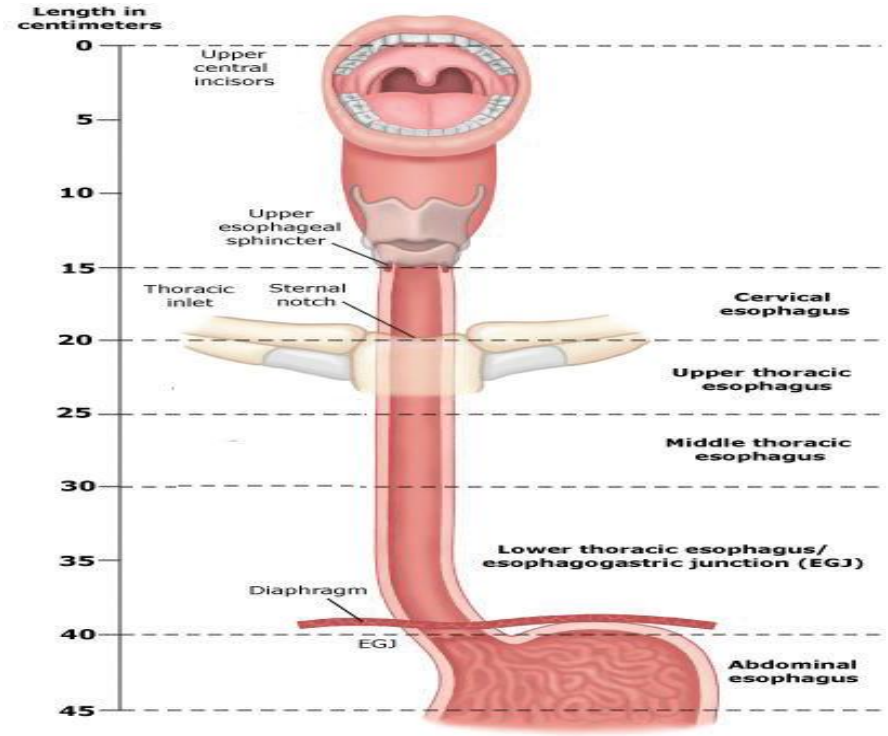

Fig. 1: Length of esophageal parts ***(Minkari et al., 1980).***