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The Role of Laparoscopic Anti-Reflux Surgery in Obese Patients with Gastroesophageal Reflux Disease

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Abstract

There has been an important increase in the prevalence of GERD in the United States in the last two decades and obesity seems to play an important role. GERD and its symptoms occur as result of anatomic and physiologic abnormalities. Pathologic reflux is a consequence of a disruption of the normal anti-reflux barrier of the lower esophageal sphincter, but it can also be a result of factors that increase the normal pressure gradient between the abdomen and thorax (for example obesity and pregnancy), esophageal motility disorders, diaphragmatic hiatal muscles and/or the stomach.

Obesity is an important risk factor for the development of GERD. Compared with patients with a normal weight, obese patients present an increased intra-abdominal pressure. These patients are a therapeutic challenge for surgeons since decreases in the Lower esophageal sphincter pressure and the presence of more frequent TLESRs are more common in this subset of patients. Even though laparoscopic surgery has been proven safe for obese patients, surgical literature is non-conclusive and its capacity to achieve a long term control of symptoms in patients with GERD it is still a matter of debate.

An obese patient considered for a laparoscopic fundoplication should undergo a careful clinical evaluation. Offering a bariatric surgical procedure instead of a traditional laparoscopic fundoplication may increase the chances of reflux symptoms relief as well as a metabolic improvement.

Abbreviations

GERD: Gastroesophageal Reflux Disease; RYGBP: Roux-en-Y Gastric Bypass; LES: Lower Esophageal Sphincter; UES: Upper Esophageal Sphincter; TLESR: Transient Lower Esophageal Sphincter

Introduction

Mechanisms of pathologic gastroesophageal reflux

GERD is a commonly treated issue by primary care physicians. About 20% of Americans experience this disease. About 14 billion dollars are spent in the management of this problem (60% on medications alone). Even though pathogenesis of GERD is not fully understood, pyrosis is believed to be caused by activation and stimulation of distal esophageal chemoreceptors. Pain associated with pyrosis is usually secondary to gastric acid exposure but it can also be caused by direct injury from biliary salts in the lower esophagus, esophageal distention and distal motor esophageal dysmotility disorders. There has been an important increase in the prevalence of GERD in the United States in the last two decades and obesity seems to play an important role. The availability of endoscopic surveillance makes it an important

contributor in detecting new cases. GERD and its symptoms occur as a final result of anatomic and physiologic abnormalities. Under normal circumstances there exists positive intra-abdominal pressure and negative intra-thoracic pressure which promotes acid reflux into the esophagus. Pathologic reflux is a consequence of a disruption of the normal anti-reflux barrier of the lower esophageal sphincter, but it can also be a result of factors

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that increase the normal pressure gradient between the abdomen and thorax (for example obesity and pregnancy), esophageal motility disorders, diaphragmatic hiatal muscles and/or the stomach [1].

The distal esophagus and LES are dynamic and closely related. The esophageal anti-reflux mechanism consists of the LES, the angle of His and the diaphragm muscle fibers. The LES measures about 2 to 4 cm long and the diaphragmatic hiatus is composed of a circular smooth muscle layer. Gastroesophageal reflux occurs when inappropriate relaxation of the LES allows the passage of gastric acid into the distal esophagus, stimulating chemoreceptors causing irritation and symptoms. The LES is a smooth muscle layer within the distal esophagus that generates a resting pressure which is normally higher than intra-abdominal pressure [2]. The right and left crus of the diaphragm compose the second defense mechanism that protects the esophagus from acid reflux. The phrenoesophageal membrane connects the crural muscles with the LES. The swallowing mechanism propels the bolus from the pharynx to the esophagus and finally to the stomach. This process can be initiated as a conscious action or as an involuntary reflex by stimulation in the mouth or pharynx. The esophageal phase of the swallowing mechanism moves food through the esophagus into the stomach against a resting pressure gradient of 12 mmHg (-6 mmHg in the thoracic cavity to +6 mmHg in the abdomen). The UES rapidly closes after the swallowing mechanism starts and sequential contractions alternating with proximal relaxations promote the bolus downwards on a peristaltic wave manner [3].

The resulting symptoms of GERD are caused by injury to the esophageal mucosa and are directly related to the number of reflux episodes, duration of acid exposure and the injury potency of the reflux content. The main mechanism which promotes reflux is named Transient Lower Esophageal Sphincter Relaxations which are activated by different stimuli such as gastric distention. An exacerbation of this effect is commonly seen in patients with hiatal hernia whom experience significant pyrosis [4]. The disruption between the crural muscle and phrenoesophageal membrane secondary to a hiatal hernia creates a proximal pouch in the distal esophagus. This has been named acid pouch and can create an environment with increased acid exposure [5]. The development of a hiatal hernia is not well understood. Nevertheless, this is usually seen more frequently in obese patients. A hiatal hernia of more than 2 cm is associated with severe esophagitis and Barrett's esophagus. Repeated shortening of the esophagus as a result of swallowing and the loss of elasticity of the phrenoesophageal ligaments are the main reasons to develop a hiatal hernia [6]. In type I hiatal hernias a circumferential weakening of the phrenoesopha-

geal ligament promotes a migration of the gastroesophageal junction proximally into the lower mediastinum. Type II hiatal hernia (Paraesophageal hernias) are a result of local weakening of the lateral phrenoesophageal ligament resulting in migration of the gastric fundus up to the lower mediastinum [2]. The third component of GERD is an abnormality in gastric function. A delay in gastric emptying can cause prolonged retention of food into the stomach which increases propensity to GERD symptoms (because there is also an increase in the pressure gradient) [5].

Discussion

Laparoscopic anti-reflux surgical treatment in obese patients with GERD

Patients with acid reflux or non-acid reflux which fail to improve on conservative management (medications such as proton pump inhibitors) may benefit from surgical procedures [1]. Anti-reflux surgery should be offered if medical therapy fails or if patients are non-compliant with medications intake, also if they present complications from acid reflux such as reflux induced asthma, aspiration pneumonia or laryngitis. Pyrosis and regurgitation are the most common symptoms in GERD patients [7].

Obesity is an important risk factor for the development of GERD. Compared with patients with a normal weight, obese patients present an increased intra-abdominal pressure. These patients are a therapeutic challenge for surgeons since decreases in the Lower esophageal sphincter pressure and the presence of more frequent TLESRs are more common in this subset of patients [8]. Even though laparoscopic surgery has been proven safe for obese patients, surgical literature is non-conclusive and its capacity to achieve a long term control of symptoms in patients with GERD it is still a matter of debate [9]. Selected patients may benefit from laparoscopic gastric bypass with Roux-en-Y reconstruction. This method has more duration of weight loss and control of comorbidities related to obesity including GERD. In morbidly obese patients and GERD a RYGBP has to be always considered instead of a typical laparoscopic fundoplication. The final decision between choosing RYGBP and laparoscopic fundoplication is based on a careful evaluation of the patient and the additional benefits of a bariatric procedure [10]. Surgical procedures aimed for obese patients that may actually work in reducing GERD symptoms include RYGBP, adjustable gastric band, laparoscopic sleeve gastrectomy and biliopancreatic diversion with duodenal switch. Nevertheless, in non-obese patients, laparoscopic fundoplication remains as the gold standard for GERD treatment [8].

There is an increased index of laparoscopic fundoplication failure in obese patients and many of them require a conversion to bariatric procedure which becomes technically more difficult and present a raised index of complications [11]. There are different factors that produce an increased pressure gradient of the gastroesophageal junction in obese patients which include the mechanical separation between the LES and extrinsic compression of the crus of the diaphragm which is an essential factor in the formation of hiatal hernias. Functional results after laparoscopic fundoplication in obese patients are still a matter of debate in surgical literature [12]. The author studied 210 patients whom underwent laparoscopic fundoplication, and the results showed that obese patients (BMI ≥ 30) were 2.5 times more likely to present laparoscopic fundoplication failure compared with non-obese patients [13]. Akimoto, et al. reported a similar result showing that obese patients have twice the chance of symptomatic GERD compared with normal population and an increase in BMI as an associated factor for increased acid exposure [14].

Laparoscopic fundoplication has been the mainstay of therapy in advanced GERD and the satisfaction rate is close to 90% at 10 years in non-obese patients [15].

Many factors have been implicated as risk factors in fundoplication failure. Nevertheless, obesity alone has been related to an increased laparoscopic surgical failure rate and an increase on intra-abdominal pressure as the main reason [16]. On the other hand, D'Alesso, et al. reported that fundoplication results were similar between BMI categories [17]. Generally, a BMI of ≥ 35 has been used as a cutoff point to decide on making a gastric bypass as both a bariatric and anti-reflux procedure in obese patients [18]. Gastric bypass has been considered the best therapeutic option for patients whom present surgical failure and need a redo procedure. This also true for non-obese patients as well [2].

Pérez, et al. Reported that up to 31.3% of obese patients presented reflux symptoms after a laparoscopic fundoplication and this result was greater than in general population [13,16]. The incidence of hiatal hernia is significantly higher in morbidly obese patients compared with non-obese patients. It is unknown if this failure rates can be attributed to an increase of intra-abdominal pressure or a poor surgical technique [14]. A bariatric procedure (specifically a RYGBP) is preferred for patients with these signs: 1) Obese patients with a BMI of 35 or more, 2) Severe fibrosis that may complicate a redo procedure, 3) Past surgical history of more than one redo procedure, 4) Evidence of pathologic tissue that requires gastroesophageal resection, 5) Severe esophageal dysmotility, 6) Slow gastric emptying and 7) Esophageal

shortening [19]. The anatomical surgical failure patterns are similar in obese and morbidly obese patients and significantly different in patients with a normal weight [14].

Conclusion

An obese patient considered for a laparoscopic fundoplication should undergo a careful clinical evaluation. Offering a bariatric surgical procedure instead of a traditional laparoscopic fundoplication may increase the chances of reflux symptoms relief as well as a metabolic improvement. Nevertheless, a skilled surgeon should be the one to perform this kind of advanced laparoscopic surgeries since technical demand and associated complications may go beyond a general surgeon's expertise.

Conflicts of Interests

The authors declare no conflicts of interests.

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