Techniques in Gastrointestinal Endoscopy 17 (2015) 83–88

Contents lists available at ScienceDirect

Techniques in Gastrointestinal Endoscopy

journal homepage: www.techgiendoscopy.com/locate/tgie

Gastroesophageal reflux disease in the obese

Diego R. Camacho, MD*, Patrick E. LeMasters, MD

Montefiore Medical Center Albert Einstein College of Medicine, 111 East 210th St Bronx, New York, New York 10467

ARTICLE INFO

Article history:
Received 2 February 2015
Accepted 22 February 2015

Keywords:
Gastric bypass
Sleeve gastrectomy
Adjustable gastric band
Weight loss
Lower esophageal sphincter

ABSTRACT

Obesity and gastroesophageal reflux disease (GERD) are common conditions that both have been on the rise. The direct relationship between GERD symptoms and obesity has been demonstrated in many epidemiologic studies. The pathophysiological mechanisms that cause this relationship have also been proposed and include decreased pressure of the lower esophageal sphincter, increased episodes of transient lower esophageal sphincter relaxations, increased intra-abdominal and intragastric pressures, and hiatal hernia. Medical treatment with either weight loss or proton pump inhibitors is effective at treating GERD symptoms in the obese. Antireflux surgery in patients with elevated body mass index has been described with varied results. Bariatric procedures have also been evaluated for their effect on GERD, with laparoscopic gastric bypass offering the best results when compared with laparoscopic sleeve gastrectomy or laparoscopic adjustable gastric banding. Those with GERD and obesity may benefit from bariatric surgery, and referral for it should be considered.

1. Introduction

The prevalence of gastroesophageal reflux disease (GERD) and obesity and the incidence of their complications have risen strikingly over the past few decades, leading to substantial economic effect [1]. Symptoms of GERD seem to be more common now than 25 years ago. In systematic reviews of population-based studies, evidence suggests an increase in GERD prevalence since 1995, particularly in North America [2]. The prevalence of GERD in North America is currently 18%-27.8% [2]. Similarly, the rate of obesity, defined as a body mass index (BMI) >30, has risen markedly with almost 33% of adults in the United States being obese [1]. Furthermore, in obese individuals, the prevalence of GERD is considered substantially higher than the general population [3].

With this rise in obesity and GERD, today's physician is faced with the challenge of how to best manage these patients. In this article, we review the epidemiology, pathophysiology, and medical and surgical management of patients with both GERD and obesity.

2. Epidemiology

The relationship between obesity and GERD has been the subject of numerous studies over the past 20 years. They show mixed reports regarding causality, but the overwhelming majority of studies show a correlation between increasing BMI and increased GERD symptoms. This includes several epidemiologic studies that clearly have established a connection [4].

In a study of 10,545 women, there was an observed dose-dependent relationship between increasing BMI and frequent reflux symptoms such that the odds ratio (OR) for GERD in a patient with a BMI of 35 or more was 2.93 (95% CI: 2.24-3.85). Also, in women with a normal baseline BMI, an increase in BMI of more than 3.5, as compared with no weight changes, was associated with an increased risk of frequent symptoms of reflux with an OR of 2.80 (95% CI: 1.63-4.82) [4].

In a cross-sectional study from Norway of 65,653 people, there was a dose-response association between increasing BMI and reflux symptoms in both sexes, with a significantly stronger association in women. Compared with those with a BMI less than 25, the risk of reflux was increased significantly among severely obese (BMI $=35$) men (OR $=3.3$; 95% CI: 2.4-4.7) and women (OR $=6.3$; 95% CI: 4.9-8.0). Reduction in BMI was associated with decreased risk of reflux symptoms in this study [5].

Similarly, El-Serag et al analyzed 453 VA employee volunteers and also demonstrated a direct relationship between BMI, GERD, and esophagitis. A dose-response relationship between frequency of GERD and higher BMI was observed such that obese participants were 2.5 times as likely as those with normal BMI (<25) to have reflux symptoms or esophageal erosions [6].

These studies mainly used questionnaires to show the correlation between GERD and BMI. Some studies have demonstrated that obesity correlates with increased esophageal acid exposure. In

The author reports no direct financial interests that might pose a conflict of interest in connection with the submitted manuscript.

* Corresponding author.
E-mail address: dicamach@montefiore.org (D.R. Camacho).

http://dx.doi.org/10.1016/j.tgie.2015.02.009
0049-0172/

2008, Crowell et al evaluated 157 patients separated into groups according to BMI. They were studied for 48 hours while off antisecretory medications using ambulatory wireless pH-metry. Obese patients had a 5-fold increase in odds for abnormal total acid exposure compared with normal-weight individuals (OR = 5.01; 95% CI: 2.94-12.95). Total acid exposure time was elevated in the obese (8.7% ± 5.1%) compared with normal-weight (5.3% ± 5.2%; $P < 0.05$) individuals. The DeMeester score was higher in the obese (31.7 ± 19.2) and overweight (26.0 ± 16.8) groups compared with the normal-weight (19.8 ± 17.6) group ($P < 0.001$) [7].

3. Pathophysiology

There is no defined pathophysiological cause for GERD in the obese, but many different factors have been described. These mechanisms have included diminished lower esophageal sphincter (LES) pressure, increased frequency of transient LES relaxations, increased intragastric pressure, increased gastroesophageal pressure gradient, esophageal and gastric motility disorders, and the presence of hiatal hernia [8].

Increased acid exposure in morbidly obese patients was shown by El-Serag et al when they compared patients with BMI greater than 30 with those less than 25. They found that those with BMI greater than 30 had significantly more total acid reflux episodes and time with pH < 4 [9]. Similarly, it has been found that patients that are overweight have significantly higher distal esophageal acid exposure when compared with normal-weight subjects [10].

In a study of 345 patients undergoing preoperative workup for bariatric surgery that completed an upper endoscopy, 24-hour pH monitoring, manometry, and reflux symptom questionnaire, 35.8% reported reflux symptoms, hiatal hernia was present in 58%, and decreased LES pressure was found in 17.7% [11].

In 2013, Anggiansah et al reported on 582 patients with typical reflux symptoms that were studied with manometry, 24-hour pH studies, and symptom severity questionnaires, and the association between obesity, esophageal function, acid exposure, and reflux symptoms was assessed. Esophageal acid exposure increased with obesity and was associated with LES pressure, reduced abdominal esophageal length, and peristaltic dysfunction. Reflux symptoms increased with acid exposure ($R = 0.300$; $P < 0.001$), and this association explained increased symptom severity in obese patients [12].

In contrast to BMI or waist circumference, abdominal visceral adipose tissue volume may be associated with an increased risk of erosive esophagitis. Nam et al in 2010 reported on 5329 patients with a prevalence of erosive esophagitis in 9.3%. The multivariate OR for erosive esophagitis was 1.97 (95% CI: 1.34-2.90) for a visceral adipose tissue volume of 500-999 cm^3, 2.27 (95% CI: 1.51-3.39) for 1000-1499 cm^3, and 2.94 (95% CI: 1.87-4.62) for ≥ 1500 cm^3, compared with participants who had visceral adipose tissue volumes less than 500 cm^3 [13]. However, others concluded that visceral adipose tissue did not correlate with BMI in morbidly obese patients and LES pressures and that GERD-related symptoms were not dependent on visceral adipose tissue [14].

Compared with normal subjects, patients with morbid obesity and those with GERD show a substantial increase in transient LES relaxations in the postprandial phase [15]. Abnormal postprandial LES function may be an early event in the pathogenesis of GERD in the obese, as it has been shown that they have transient LES relaxation in even those without GERD [16]. In 2009, 1659 patients referred for the assessment of GERD symptoms underwent 24-hour pH monitoring off medication and esophageal manometry. The relationship of BMI to 24-hour esophageal pH measurements and LES status was analyzed. The difference of each acid exposure component was also assessed among BMI subgroups from underweight to obese. Increasing BMI was positively correlated with increasing esophageal acid exposure. In addition, compared with normal-weight patients, obese patients were more than twice as likely to have a mechanically defective LES (OR = 2.12 [1.63-2.75]) [17].

Furthermore, in a study of 287 patients by Pandolfino et al, performed to determine the relationship of obesity and the morphology of the esophagogastric junction pressure segment using high resolution manometry, there was a significant correlation of both BMI and waist circumference with intragastric pressure and gastroesophageal pressure gradient. They concluded that obese subject are more likely to have esophagogastric junction disruption leading to hiatal hernia and an augmented gastroesophageal pressure gradient providing a perfect scenario for reflux to occur [18].

In a more recent study, 245 patients that were normal weight, overweight, and obese were study with manometry, 24-hour pH-metry, and upper endoscopy. Overweight and obese patients had increased reflux, especially in the supine position. The most important factors that contributed to reflux were the presence of a hiatal hernia and a lower LES pressure in overweight and obese patients as well as an increased intragastric pressure. A hiatal hernia was significantly more often present in overweight and obese patients ($P = 0.01$) compared with the normal-weight group [19].

Other reports show that reflux disease is mediated through a rise in the intra-abdominal pressure and distortion of the gastroesophageal junction [20]. Derakhshan et al compared 103 obese patients with 18 healthy volunteers who had an abdominal belt applied. There was a linear correlation between BMI and esophageal acid exposure. BMI was strongly associated with increased intragastric pressures. Application of the constricting abdominal belt on healthy volunteers produced similar manometric changes to those with increased BMI. This suggests the association with reflux and increased BMI may be partially explained by increased intra-abdominal pressure [21].

Hiatal hernia is commonly associated with symptomatic GERD, and patients with abnormal esophageal acid exposure have a higher prevalence of hiatal hernia. The prevalence of hiatal hernia (37%) and GERD (39%) was found to be very high in patients undergoing routine upper gastrointestinal (UGI) studies before bariatric surgery [22]. The risk of reflux was also predicted in severely obese patients (BMI = 45) by the presence of hiatal hernia with a prevalence ratio of 1.15 (CI: 1.19-2.00; $P = 0.001$) [23].

4. Therapeutics

4.1. Medical management

With the overwhelming evidence of how obesity affects GERD, the obvious first-line intervention to propose would be medical weight loss. In total, 332 adult patients with a mean BMI of 35 underwent an intensive weight loss regimen over 6 months and had a change in GERD prevalence from 37%-15%. Overall, 81% of the subjects had reduction in GERD symptom scores and 65% had complete resolution. Furthermore, there was a significant correlation between percent weight loss and reduction in GERD symptom scores [24]. In a study of contributing lifestyle measures, weight loss and head-of-the-bed elevations were the 2 interventions with supporting evidence of benefit. Cessation of tobacco, alcohol, and other dietary interventions had no evidence for benefit [25].

Proton pump inhibitors (PPI) have been the mainstay of treatment for GERD. There are limited data describing PPI response