

ADVANCES IN GERD

Current Developments in the Management of Acid-Related GI Disorders

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Gastroesophageal Reflux and the Obesity Epidemic

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G&H Could you characterize the current obesity epidemic as it relates to the US populace?

HE Current data indicate that up to two thirds of adult Americans are overweight and one quarter of Americans are obese. Even more disturbing is the rapidly rising incidence of obesity in children and adolescents. The obesity epidemic is likely caused by increased food intake (both increased frequency and portion size), increased availability, increased advertisement, and increased intake of high-calorie foods. In addition, energy expenditure continues to decrease, with less exercise, less physical activity, more sedentary work, and more sedentary entertainment. This last factor is particularly true in children, who partake increasingly in electronic entertainment rather than outdoor activities.

G&H How has the association between obesity and GERD been documented?

HE Obesity affects several aspects of gastroesophageal reflux disease (GERD). It has been linked to increased frequency and severity of GERD symptoms as well as increased prevalence of erosive esophagitis. Several population-based studies from different countries have shown that obesity increases the risk of esophageal adenocarcinoma by a two- to three-fold magnitude. Lastly, obesity is possibly associated with increased risk of Barrett esophagus although the evidence for this association is sparse.

Whether these causal associations are confounded by other factors, like dietary intake and/or physical activity, is not clear. The few studies that have been performed

adjusting for dietary factors found that obesity remained an independent risk factor, but this topic requires further examination. Another question is whether overall obesity or abdominal obesity is the determining factor in causing GERD. Preliminary evidence shows that abdominal obesity, as indicated by waist circumference, is a stronger risk factor than overall obesity reflected by body mass index (BMI).

Several studies have indicated that obese persons, particularly those with a larger waist circumference, have increased esophageal acid exposure, lower esophageal sphincter pressure, and a high gastroesophageal pressure gradient, which facilitate free reflux and possibly increased frequency of transient relaxation of the lower esophageal sphincter. These studies show that the obesity causality theory is biologically plausible because these mechanistic changes seem to be associated with obesity itself. There are also uncontrolled studies of obese bariatric surgical patients presenting with symptoms of reflux or esophagitis who report dramatic improvement following the surgery. However, there are no controlled trials to confirm this finding.

G&H Can loss of weight with exercise and dietary modification reduce GERD symptoms?

HE The most appropriate interpretation of current evidence is that avoiding initial excessive weight gain is the best way to prevent reflux. Whether reducing weight will reduce risk is not clear. Particularly in instances where an irreversible change manifests, such as hiatal hernia or Barrett esophagus, the risk of complications will not be reduced. Therefore, I believe the best advice is to avoid gaining weight initially. In regard to diet itself, high-fat and low-fiber foods have been implicated in increased risk of reflux symptoms as well as erosive esophagitis and esophageal adenocarcinoma.

G&H What other factors contribute to the increased prevalence of GERD, independent of weight gain?

HE One epidemiologic hypothesis is that the declining prevalence of *Helicobacter pylori* infection in the general population has been associated with improved gastric-acid-secretion capacity. Therefore, if the predisposing conditions to GERD are present, the refluxate is more injurious. Another risk factor that may contribute to GERD is increased use of medications that may interfere with lower esophageal sphincter relaxation, such as calcium-channel blockers, nitrates, and beta blockers. Lastly, there is an increased awareness of the disorder, which can result in increased detection rather than a true increase in prevalence. However, if indicators of GERD such as esophageal adenocarcinoma are examined, there is still an increase that cannot be explained by increased awareness.

G&H Would you describe your own meta-analysis on the link between BMI and GERD?

HE Our group reviewed published literature from 1966 through October 2004 and found nine studies dealing with the association between BMI and the major manifestations of reflux—weekly symptoms, endoscopically-defined erosive esophagitis, Barrett esophagus, and esophageal adenocarcinoma. By pooling the results from the studies, we found a one- to two-fold increase in the risk of GERD symptoms associated with obesity and a two- to three-fold increase in the risk of adenocarcinoma. At the time of the meta-analysis, there were no eligible studies of the association between obesity and Barrett esophagus. This is still the most understudied area. We also found a dose-response relationship where obesity, as defined by BMI calculated as greater than 30, carried a higher risk of GERD than overweight, defined as a BMI of 25–30. This showed a relationship of more weight to more risk.

G&H Have you conducted any additional research on this topic?

HE Our group also conducted a single-center retrospective study of patients who underwent endoscopic examination and were either diagnosed with Barrett esophagus or had no Barrett-related findings. Patients in both groups also had received an abdominal computed tomography (CT) scan (for any reason) within a year of the endoscopy. We examined the stored electronic records of the CT scans using software to calculate the surface area of different fat compartments and found that the surface area of visceral abdominal fat is the most important risk factor for Barrett esophagus, whereas the amount of subcutaneous fat was not an important risk factor. If these findings can be confirmed in large, prospective studies, the implication may be that not only the volume but also the distribution of fat in the abdomen is important. It is known that visceral fat is an active metabolic organ that produces a variety of adipocytokines, including interleukin-1 and -6 and tumor necrosis factor. Some of these adipocytokines increase inflammation throughout the body, including the esophagus. Thus these findings may open an area of research that addresses a humoral mechanism for obesity-related reflux, in addition to the reasonably well-established mechanical aspects of abdominal obesity.

Suggested Reading

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