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OBESITY MANAGEMENT INTERVENTIONS AND THEIR IMPACT ON CLINICAL INDICATORS OF GERD

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Abstract: Gastroesophageal reflux disease (GERD) and obesity are two highly prevalent chronic conditions that share multiple pathophysiological mechanisms, including increased intra-abdominal pressure, lower esophageal sphincter dysfunction, and systemic inflammation. Recent studies suggest that effective obesity management may contribute to the improvement of GERD symptoms and reduce the risk of esophageal complications. This review aims to evaluate the impact of different obesity treatment strategies such as lifestyle modification, pharmacotherapy, and bariatric surgery on the clinical course, symptom severity, and objective diagnostic indicators of GERD.

Key words: Obesity, gastroesophageal reflux disease, fundoplication, sleeve gastrectomy, gastric banding, gastric bypass.

Introduction. Gastroesophageal reflux disease (GERD) is a frequently encountered disorder. Obesity is an important risk factor for GERD, and there are several pathophysiologic mechanisms linking the two conditions. For obese patients with GERD, much of the treatment effort is focused on weight loss and its consistent benefit to symptoms, while there is a relative lack of evidence regarding outcomes after novel or even standard medical therapy is offered to this population. Physicians are hesitant to recommend operative anti-reflux therapy to obese patients due to the potentially higher risks and decreased efficacy, and these patients instead are often considered for bariatric surgery. An important risk factor for GERD is obesity, which has been increasing in prevalence and is strongly associated with adverse metabolic, cardiovascular, chronic inflammatory and malignant health outcomes. Notably, the treatment of obesity remains at the forefront of preventative healthcare.

A variety of non-surgical and surgical obesity treatments can impact the severity and natural history of its associated diseases. This article aims to review the evidence surrounding the epidemiology, pathophysiology and potential impact of obesity treatment on GERD.Although it has limitations the degree of adiposity in adults can be categorized by the body mass index (BMI) calculation (weight in kilograms divided by height in meters squared). Both overweight (BMI 25-29.9) and obese (BMI ≥ 30) patients have been shown to be at higher risk for GERD. The Nurses' Health Study data demonstrated that incremental weight gain among patients with a normal BMI (18-24.9) is associated with a proportional increased risk for reflux symptoms, whereas weight loss was associated with decreased reflux symptoms. Specifically, the risk of reflux symptoms was linked to the parameter waist- to-hip ratio in a dose-responsive fashion, though BMI itself appeared to be even more closely associated with symptoms than this ratio. Several mechanisms have been

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postulated to link obesity and the development of GERD. One area of investigation has focused on the anatomic displacement of the esophagus into the chest cavity.

Obese patients are over three times as likely to have hiatal hernias compared to non-obese individuals. Furthermore, after controlling for hiatal hernias, the relationship between BMI and esophagitis was non- significant in one study, suggesting that hiatal hernias mediate the link between obesity and GERD. Hiatal hernias have been linked to obesity-associated reflux symptoms due to the increased intragastric pressure and gastro-esophageal pressure gradients during inspiration, as well as increased axial separation between the extrinsic crural diaphragm and the lower esophageal sphincter (LES) in the setting of an elevated BMI. Extrinsic gastric compression from increased visceral adiposity in overweight and obese patients may also lead to increased intragastric pressure and thus a favorable pressure gradient for reflux to occur. Obese patients have been reported to have higher rates of esophageal motility disorders and bolus transit impairments compared to normal BMI patients with GERD. Mechanisms leading to an increased prevalence of GERD in obese patients may also differ according to gender. Although questionnaire-based studies have reported similar GERD symptoms between men and women, one study using pH monitoring reported that women secrete less gastric acid and tend to have less physiologic reflux compared to men.

The effect of estrogen on GERD has been examined, as estrogen increases the synthesis of nitric oxide, a vasodilator leading to smooth muscle relaxation that can include the LES. The effect of hormone replacement therapy (HRT) on GERD has been analyzed in several studies, specifically estrogen versus combined estrogen and progesterone. Historically, progesterone treatment was linked to GERD, but recently it was demonstrated that estrogen only HRT was more strongly associated with GERD symptoms than was combined estrogen and progesterone treatment. However, when analyzing the impact of naturally occurring sex hormones on GERD among individuals not on HRT, it was found that sex hormone levels in both pre- and post-menopausal women and excess acid exposure were not associated once age and BMI were taken into account. The authors in this study concluded that another mechanism, such as increased intragastric pressure associated with a higher BMI, better explains the sex hormone-BMI- GERD dynamic. The authors further concluded that the increased rates of GERD in pregnant patients may in fact not be due to increased sex hormone levels but rather the increased transmitted gastric pressure from the enlarged uterus.While fewer studies have looked at differences among ethnicities, Corley and authors found that the correlation between BMI and reflux symptoms was highest in whites and lower in blacks or Asians. The authors suggested that differential patterns of visceral adiposity among ethnicities along with differences in Helicobacter pylori prevalence could also explain the findings.

Adipokines have also been an important area for research on GERD and obesity, with increasing interest in ghrelin and leptin. Ghrelin, an orexigenic hormone, acts in the arcuate nucleus of the hypothalamus by activating neuropeptide Y (NPY) and agouti-related peptide (AgRP) neurons, leading to increased food intake. The adipose tissue-derived hormone leptin is produced in proportion to fat stores. Circulating leptin serves to communicate the state of body energy repletion to the central nervous system in order to suppress food intake and permit energy expenditure.In a study comparing rats with surgically-induced GERD to those with sham operations, the rats with GERD had significantly decreased food intake and body weight, despite having significantly increased levels of ghrelin. Even with repeated administration of acyl ghrelin, GERD rats had no improvement in food intake or body weight, suggesting that GERD rats may need a higher level of exogenous ghrelin for its predicted effect. The authors concluded that aberrantly

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increased secretion of peripheral ghrelin and decreased ghrelin responsiveness may occur in GERD rats. When analyzing the various steps in the orexigenic signaling pathway, the authors also found that while NPY and AgRP mRNA expression in GERD rats was increased, the GERD rats appeared to have dysfunction further along in the orexigenic signaling pathway, potentially leading to suppression of ghrelin signaling. In humans ghrelin has been reported to be positively associated with Barrett's esophagus but inversely associated with GERD symptoms.

Leptin has also been implicated in the relationship between obesity and GERD symptoms. In a study on Egyptian patients with GERD, leptin was found to be significantly positively correlated with symptom score severity, weight, BMI, waist circumference, waist- to-height ratio, total abdominal fat, subcutaneous abdominal adipose tissue, and intra-abdominal adipose tissue; yet it was negatively correlated with waist-to- hip ratio.

The adipokine adiponectin has been investigated in GERD, because it is secreted from visceral fat and can suppress tissue inflammation. Adiponectin has been shown to be inversely correlated to BMI, and in a study on male patients with and without erosive esophagitis, serum adiponectin levels were found to be lower in patients with erosive disease. The authors concluded that low adiponectin levels may be associated with an increased risk for erosive esophagitis in men.

A recent study has also shown a link between nonalcoholic fatty liver disease (NAFLD) and GERD. While NAFLD has a known association with obesity, in this study of 206 outpatients with NAFLD in a single center the prevalence of typical GERD symptoms was higher in these patients compared with controls and independent of BMI. The authors postulated that higher serum levels of certain cytokines found in patients with NAFLD, which can be independent of obesity and also overexpressed in the esophageal mucosa of GERD patients, may help explain the findings. As NAFLD has been proposed as a potential new criterion to define metabolic syndrome and is associated with many of the outcomes of obesity, further study on the specific role in NAFLD leading to GERD is warranted.Overall, while there are several pathophysiologic mechanisms that may explain the risk of GERD in the obese population, both the predisposition for hiatal hernias and direct impact of visceral adiposity remain the most substantiated. Further studies are needed to decipher the influence of adipokines on the development of GERD in this population, as well as the potential differences in the incidence of GERD by gender and ethnicity.Lifestyle modifications have long been paramount to the management of GERD, and while most behavioral factors have not been extensively studied in the obese population, many of the interventions have proven to be effective in both normal weight and obese individuals.

Weight gain and weight loss are associated with an increase and decrease in reflux symptoms, respectively, in both normal and overweight individuals. One study specifically demonstrated that weight loss via dietary advice can improve reflux symptoms in overweight individuals.While not specific to obese individuals, elevation of the head of the bed, left lateral decubitus position, and deep breathing exercises (inducing a change from thoracic to abdominal breathing) are associated with improvement of GERD symptoms. Furthermore, both fruit and fiber consumption in the overall population have been found to be protective against GERD symptoms, yet whether this effect is independent of a patient's BMI has not been evaluated.Coffee and caffeine have also been examined for their effects on GERD symptoms. One study has shown that coffee, independent of caffeine, itself may be responsible for the reflux symptoms. Further complicating the potential relationship between coffee, caffeine and a predilection for GERD are two discordant studies showing that coffee either

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can lower the LES pressure (LESP) or increase it. The recommendation to avoid caffeinated beverages is still used routinely in patients with GERD, but whether coffee or caffeine itself is a major factor in the pathophysiology of GERD, particularly in the obese population, remains to be elucidated.The effect of protein and fat has been studied in both the general and obese population. Protein and dietary fat have been found to have opposite effects on the LES; ingestion of protein increased LESP whereas ingestion of fat decreased LESP. Ingestion of total fat, saturated fat, and cholesterol were found to be higher in patients with GERD symptoms than those without symptoms in one study. However, only in patients with a BMI > 25 were high saturated fat, cholesterol, and fat servings associated with GERD symptoms. Dietary fat may have an independent role in the pathophysiology of GERD, particularly in obese people.

In addition to lifestyle changes, a variety of medications are used in the management of GERD. Particularly, antacids, H2 receptor antagonists (H2RAs) and proton pump inhibitors (PPIs) are often necessary to provide as acid inhibition. McDougall et al found an association between higher BMI and the requirement of longer-term H2RA or antacid therapy, suggesting that obese patients are not as responsive to these medical treatments. The efficacy of PPIs in obese patients with GERD was not affected by BMI in one study, however, another report noted that doubling the dosage of pantoprazole in obese or overweight patients provided better specific control of symptoms, and other studies have corroborated these findings. Novel treatments, including CCK inhibitors, GABA agonists, and TCAs are currently being studied for their effects on GERD symptoms, but there is a lack of data on their precise roles in the obese population.

CONCLUSION

Obesity and GERD have a well-defined association due to several anatomic and hormonal pathophysiologic mechanisms. Ultimately, while the medical and surgical treatment of GERD is advancing, there is a relative lack of specific studies looking at novel GERD treatments in the obese population. The primary focus in these patients still remains the reversal of the excess weight, either with lifestyle modification or consideration of bariatric surgery.

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