

Esophageal Carcinoma Following Bariatric Procedures

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ABSTRACT

Background: The long-term success of bariatric operations for weight reduction has been well documented, but their potential effects on the risk of esophageal cancer have not been evaluated.

Methods: We performed operations on 3 patients for esophageal cancer following bariatric operations: 2 had Roux-en-Y gastric bypass, and 1 underwent vertical banded gastroplasty. All of these patients had adenocarcinoma at the gastroesophageal junction; 1 involved the entire intrathoracic esophagus.

Results: The intervals between the weight-loss operations and cancer diagnoses were 21, 16, and 14 years. All 3 patients had symptoms of reflux for many years before dysphagia developed and cancer was diagnosed. We performed a limited esophagogastrectomy, a classic Ivor-Lewis procedure, and a total esophagectomy with jejunal free-tissue transfer from stomach to cervical esophagus. Two patients had positive lymph nodes. One patient is alive at 6 years; 2 died at 13 and 15 months after undergoing operation for recurrent cancer.

Conclusion: The effect of bariatric operations on gastroesophageal reflux is not known, although gastric bypass has been advocated as the "ultimate antireflux procedure." The presence of esophageal cancer in these 3 patients years after the weight loss operation is worrisome. We believe that patients who develop new symptoms should have endoscopic evaluation and that epidemiologic studies on the incidence of esophageal cancer occurring years after bariatric operation should be performed.

Key Words: Bariatric surgery, Morbid obesity, Esophageal adenocarcinoma.

INTRODUCTION

Morbid obesity [body mass index (BMI) ≥ 40 kg/m²] is reaching epidemic proportions in the United States.^{1,2} Surgical treatment of obesity, which began in the 1950s with the jejunoileal bypass, is rapidly evolving. A growing body of evidence supports the safety and efficacy of bariatric operations.³⁻⁸ Modern weight-loss operations involve some manipulation of the stomach,⁹ thereby altering the structure and function of the upper gastrointestinal tract. Adenocarcinoma of the esophagus, like morbid obesity, also has increased at an alarming rate in recent years.^{10,11} No association between weight-loss operations on the stomach and gastroesophageal cancer has been previously reported. We present herein 3 cases of esophageal adenocarcinoma that occurred after bariatric procedures and highlight the need for formal epidemiologic testing and further investigation of this phenomenon.

CASE REPORTS

Patient 1

A 54-year-old morbidly obese man underwent gastric bypass with stapled division of the stomach and Roux-en-Y gastrojejunostomy 21 years before he developed cancer. He experienced persistent symptoms of regurgitation, particularly after overeating. Upper endoscopy (EGD) disclosed a columnar-lined esophagus without atypia 5 years before his cancer was diagnosed. Surveillance endoscopy was performed on 2 occasions. Atypia had been noted 26 months earlier. The patient failed to have a follow-up EGD, but 3 months prior to presentation, he developed dysphagia. Adenocarcinoma was confirmed by EGD in biopsies from the distal, mid- and proximal esophagus. Computed tomography (CT) demonstrated a thickened esophagus with no tumor outside the chest, in the liver, or the abdomen, and no evidence of nodal involvement.

A total esophagectomy was performed with en bloc resection of the upper stomach through a midline incision and right thoracotomy. A jejunal free-tissue transfer was used for alimentary tract reconstruction, with an anastomosis between the cervical esophagus and jejunal graft

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proximally and the graft and stomach remnant distally. A tube jejunostomy was placed as well.

The specimen contained a long segment of adenocarcinoma from the gastroesophageal junction to the proximal thoracic esophagus. The patient recovered after a lengthy operative procedure and was able to eat solid food, but he required supplemental tube feedings to maintain adequate caloric intake. Cancer recurred at 8 months postesophagectomy, and he died 13 months after his operation.

Patient 2

A 31-year-old woman underwent a jejunoileal bypass in 1973 for morbid obesity. Eight years later, she had lost 64 kg of weight but was disabled by arthritis, and results of her liver function tests were abnormal. Her jejunoileal bypass was surgically reversed, resulting in prompt weight gain and improvement of her symptoms. Sixteen months later, an incisional hernia was repaired and a vertical banded gastroplasty (VBG) performed. She developed reflux symptoms that were treated with histamine receptor blocking agents. In 1999, at age 57, the patient's persistent vomiting and dysphagia necessitated performance of an upper gastrointestinal series, which disclosed a severe stenotic lesion at the gastroesophageal junction. An EGD showed an ulcerating lesion containing signet ring type adenocarcinoma.

At operation, the patient's cancer was found to extend into the lesser curvature of the stomach, which prompted a total gastrectomy. A Roux-en-Y esophagojejunostomy was performed. The patient died of recurrent intraabdominal cancer 15 months after the operation.

Patient 3

A 36-year-old man underwent a Roux-en-Y gastric bypass in 1982. He lost 80% of excess body weight, but slowly regained weight after about 10 years. Fourteen years after his gastric bypass, he developed dysphagia that was initially attributed to a stricture. An EGD showed a lesion at the gastroesophageal junction. Biopsy results were positive for adenocarcinoma. An esophagogastrectomy was performed through a combined abdominal and right thoracotomy. A gastric tube was created from the stomach, despite the previous operation on the fundus. The patient had a small primary cancer that did not invade the muscularis. The postoperative course was uneventful, and the patient remains free of recurrent cancer 6 years after his operation.

DISCUSSION

The association between bariatric procedures and subsequent development of gastroesophageal adenocarcinoma may be coincidental. However, these cases suggest a relationship between gastric weight-loss operations and the development of adenocarcinoma of the gastroesophageal junction. Although the pathogenesis of esophageal adenocarcinoma is still not fully understood,¹² these cases suggest that there may be other, unidentified etiological agents or pathophysiological processes involved.

It is possible that the gastric pouch, which represents the terminal portion of the functional stomach and is common to both gastroplasty and gastric bypass, increases exposure of the vulnerable lower esophagus to carcinogens due to their proximity. For example, compounds normally present in the gastric antrum are unavoidably placed closer to the esophagus as a result of these procedures. The link between gastric acid reflux and the development of Barrett's metaplasia, esophageal dysplasia, and adenocarcinoma is well established. The normal transit of gastric acid, secreted within the gastric pouch, is almost certainly prolonged, therefore increasing the severity and duration of acid reflux. Motility changes that can occur after the gastric manipulation required in these operations increases acid exposure.

Operations that alter the "native" contents of the gastrointestinal tract and predisposes it to cancer is not a new concept. There have been reports of cancer developing in the "neo-esophagus," following colonic interposition¹³ and Collis gastroplasty.¹⁴ Furthermore, adenocarcinoma of the jejunal component of Roux loop has also been described.¹⁵

The role of duodenogastroesophageal reflux in esophageal adenocarcinoma development has been an issue of recent debate.¹⁶⁻¹⁸ However, gastric bypass, as performed in 2 of our patients, has been advocated as the ultimate antireflux procedure¹⁷ and should, by definition, eliminate duodenogastroesophageal reflux. It has recently been suggested that a duodenal diversion (Roux-en-Y) procedure is superior to simple fundoplication in treating patients with Barrett's esophagus.¹⁹ Furthermore, several investigators have found no relationship between previous gastric surgery (as a model of duodenogastroesophageal reflux) and esophageal adenocarcinoma.^{20,21}

Much evidence exists linking adenocarcinoma of the esophagus and obesity.^{10,22-28} It is entirely possible that these patients were predisposed to esophageal cancer because of their obesity and not the bariatric procedure,

although each achieved significant weight loss following surgery. On the other hand, it is imperative that formal epidemiological research either confirm or refute this worrisome possibility. Since the cancers described above occurred many years after the bariatric operations, the original surgeon may no longer be involved in the care of such patients.

Regardless of the possible link between bariatric procedures and subsequent cancer development, clinicians must be diligent in their investigation of new symptoms that could be caused by cancer. Because many of these patients have ongoing regurgitation associated with overeating, the recognition of relevant symptoms may be problematic.

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