Risk of Upper Gastrointestinal Cancer after Bariatric Operations

Arnaud De Roover; Olivier Detry; Claude Desaive; Sylvie Maweja; Carla Coimbra; Pierre Honoré; Michel Meurisse

Department of Abdominal Surgery and Transplantation, Centre Hospitalier Universitaire de Liège, University of Liège, Liège, Belgium

Abstract

The authors discuss the potential influence of obesity surgery on the risk of cancer, focusing on the upper Gl tract directly affected by operations. There is currently no substantiation for an increased risk of cancer after bariatric surgery, because there are only about 25 reports of subsequent cancer of the esophagus and the stomach. However, this review emphasizes the need to detect potential precancerous conditions before surgery. Candidates for postoperative endoscopic surveillance may include patients >15 years after gastric surgery, but also patients symptomatic for gastroesophageal reflux disease in whom a high incidence of Barrett's metaplasia has been reported. The greatest concern is a delay in diagnosis from inadequate investigation due to mistaking serious upper Gl symptoms as a consequence of the past operation.

Keywords: Cancer, morbid obesity, bariatric surgery, Barrett's esophagus, metaplasia

Introduction

Surgery for morbid obesity is increasing in all industrialized countries and is becoming one of the most common types of surgery. The choice of operation depends primarily on the expected effect on the weight loss. A concern of the caring surgeon is to avoid harmful consequences to the patient as a result of the operation. While potential surgical and metabolic complications have been well-defined in the postoperative period, the risk of cancer associated with bariatric surgery in general and with specific operations remains unclear.

This subject is relevant from the first contact with the obese patient, because the bariatric surgeon will treat and care for a population that presents an increased risk for the development of malignancy. Increased body weight has been found to be associated with increased death rates from cancer, most consistently from endometrium, kidney, gallbladder, breast, colon and esophagus.^{1,2} In a US prospective cohort study of over 900,000 patients,³ the increased risk of obesity was identified for cancer of the esophagus, colon and rectum, liver, gallbladder, pancreas, kidney, non-Hodgkin lymphoma and multiple myeloma. In men, there was also a significant increased risk for cancer of the stomach and prostate, while in women, this risk was found for cancer of the breast, uterus, cervix and ovary. Potential etiologic mechanisms include increased levels of hormones associated with obesity (e.g. steroids, insulin, insulin-like growth factor I).

Bariatric Surgery - Rationale for Cancer Risk

Another concern rises from a potential influence of the bariatric operation on the development of cancer.

This risk has been recognized in the gastric stump after gastrectomy for benign disease (peptic ulcer surgery), with a 3-5 fold increase after 15-25 years.⁴ While the prevalence of Billroth II operations appears higher in the reported cases of gastric stump carcinoma, it may only reflect its higher frequency compared to the Billroth I technique. In addition to time since initial surgery, suggested risk factors⁵ include sex (female), age at operation (old), and underlying disease (gastric ulcer). The discrepancies in the risk reported in the literature may reflect insufficient length of follow-up in many studies, in addition to potential environmental and ethnic factors.⁶

The raised gastric pH associated with pancreatico-biliary reflux might lead to the bacterial colonization whose metabolism leads to production of carcinogens. The influence of *Helicobacter pylori* (HP) in the genesis of gastric pouch carcinoma is currently unsettled.^{4,7} The prevalence of HP after partial gastrectomy is <50% and

might be influenced by duodenogastric reflux.⁸ This bacterium has not been found to be specific in the patients presenting with dysplasia or adenocarcinoma of the gastric remnant, but in the endoscopic surveillance study after partial gastrectomy of Giuliani et al,⁹ the prevalence of intestinal metaplasia was four times higher in HP-positive patients (19.6% vs 4.6%). Ebstein-Barr virus was also suggested as a potential co-factor by Yamamoto¹⁰ et al associated with gastric remnant cancer after partial gastrectomy.

Gastric Pouch Cancer after Gastric Bypass Procedures

The above data on gastric remnant cancer after partial gastrectomy should apply to bariatric operations in which a loop jejunal anastomosis was performed on the gastric pouch (Mason loop gastric bypass). The procedure of Roux-en-Y gastric bypass (RYGBP) usually uses a long Roux loop of jejunum and should carry a different risk. This is suggested by the lesser incidence and severity of gastritis in the gastric remnant after gastrectomy for ulcer disease, if the gastric pouch is anastomosed to a Roux-en-Y loop compared to a loop gastroenterostomy.¹¹ Furthermore, atrophic gastritis which is frequent after a Billroth procedure, appears to be rare in the proximal pouch after a Roux-en-Y drainage.^{11,12}

McCarthy et al¹³ evaluated the gastric pouch of 28 patients after gastric bypass surgery according to the type of gastrojejunal anastomosis. They observed a 71% incidence of endoscopic gastritis after loop gastroenterostomy and 13% after RYGBP. Histologic examination of the mucosa demonstrated inflammation in 86% of the first group and 63% after RYGBP. Dysplasia was found in the biopsy specimens from the pouch, but their distribution according to the type of gastrointestinal anastomosis was not presented. No preoperative endoscopy had been performed, and with the short time interval between surgery and endoscopy (mean 8 months), it is difficult to ascertain the significance of the procedure in the changes observed.

Only two cases of cancer in the gastric pouch have presently been reported. One pouch adenocarcinoma occurred in a 61-year old lady 26 years after a Mason loop gastric bypass.¹⁴ *En bloc* resection included pouch, bypassed stomach, and 10 cm of transverse colon, and none of the 14 lymph nodes were involved. Reconstruction was by esophago-jejunostomy, but long-term follow up is unknown.

The other pouch carcinoma occurred in a 52-year-old lady who had initially undergone adjustable gastric banding complicated by gastric wall erosion that required band removal and conversion to RYGBP¹⁵ Five years later, she was diagnosed with a carcinoma of the pouch.

Gastric Cancer in the Bypassed Stomach after Gastric Bypass

The distal stomach after gastric bypass is also exposed to pancreaticobiliary reflux with pooled bile reported in the excluded stomach.¹⁶ Superficial gastritis of this segment is common. While Sinar et al¹⁶ reported that biopsies from the distal stomach in 4 of 33 patients showed intestinal dysplasia at 3-24 months after surgery, this was not observed in a later study with a 2 years follow-up by the same group.¹⁷ The risk of cancer of the bypassed segment cannot be derived from a similar operation and is unknown. The distal gastric segment is excluded from the alimentary channel and thus from contact with exogenous carcinogens. It could, however, suffer from prolonged contact with stagnant bile, shown experimentally to promote carcinogenesis.⁴

Five cases of adenocarcinoma of the distal stomach have been reported after gastric bypass.¹⁸⁻²² Cancer was diagnosed at an advanced stage in four of the cases, 4,5, 8 and 22 years after the procedure. The patient with the carcinoma in the bypassed stomach at 4 years had had intestinal metaplasia and HP (given eradication therapy) on endoscopy before the RYGBP.¹⁸ Symptoms resulted from distal gastric outlet obstruction in three cases and were associated with tumor infiltration of liver in the fourth patient.²¹ A fifth patient presented with anemia that was investigated for 1 year before an endoscopy evaluated the distal stomach through the loop gastrojejunostomy and found two polypoid early gastric cancers.²²

The delay in diagnosis of cancer represents a risk for lesions arising in the excluded distal stomach that are more difficult to investigate. This is illustrated in the following case referred to our surgical department:²³ A 66-year-old male 3 years after RYGBP, was diagnosed with a gastric fundus perforation associated with a diffuse large B-cell lymphoma. The patient had a 6-month history of anemia with GI bleeds, with investigated as the source of lower endoscopy having been negative. The distal excluded stomach had not been investigated as the source of bleeding, because of the absence of knowledge of the persisting distal gastric segment after bypass surgery.

Gastric *lymphoma* is a rare event, representing 3% of all gastric cancers.²⁴ In the case described above, it is uncertain whether local conditions in the bypassed stomach played a role in the development of lymphoma. The

relatively short time interval between RYGBP and malignant transformation in our patient precludes a definitive answer. Lymphomas have been described in the proximal gastric stump 5 to 43 years after partial gastrectomy, with 15 cases reported in the Western literature and 27 cases in Japan.^{25,26} The proximal gastric stump after the partial gastrectomy and the distal gastric remnant after RYGBP may share in common an increased exposure to duodenal reflux with accompanying mucosal inflammation. It has been suggested that lymphocyte infiltration associated with chronic inflammation of the mucosa could develop into lymphoma. HP also plays an important role in the development of gastric lymphoma.^{27,28} Other factors, however, intervene, as there has been a low correlation between HP and lymphoma in the reported cases after partial gastrectomy.

Gastric Cancer after Restrictive Operations

A modification of the risk of gastric cancer by a purely restrictive procedure could result from prolonged contact of food and potential exogenous carcinogens in the gastric pouch. Increased pressure with chronic inflammation in the pouch could also be evoked as a potential factor for oncogenesis that can also include irritation by the banding material. These hypotheses remain speculative in view of the few cases of cancer reported in the literature.

Four cases of adenocarcinoma have been described after vertical banded gastroplasty (VBG). Three occurred in the gastric pouch at 2, 13 and 15 years after surgery,²⁹⁻³¹ while the fourth appeared in the distal stomach 6 years after VBG³² The patients presented with a history of epigastric pain, nausea and vomiting for a few weeks to 3 months before diagnosis. Histological examination revealed a T1N0 adenocarcinoma in one case, but showed poorly differentiated adenocarcinoma with positive nodes in the other three patients, two with distant metastases.

Ten years after adjustable gastric banding, a 62-year-old lady presented with band erosion amid gastric cancer.³³ Pathologic study demonstrated a poorly differentiated adenocarcinoma with positive perigastric nodes and hepatic metastases.

The main risk of cancer associated with restrictive operations could derive from negligence of upper GI symptoms commonly observed after the operation that can delay diagnosis and treatment. This is shown in the following case report:²³ A 47-year-old lady presented in our clinic after several months of progressive dysphagia with vomiting, 12 years after a VBG. These symptoms had been attributed to inadequate respect for dietetic rules after VBG. Endoscopy and barium studies demonstrated severe stenosis with a normal mucosa at the gastric pouch outlet. At laparotomy, a 9-cm intramural mass was found in the antrum, just below the pouch outlet, and was treated by total gastrectomy. Frozen section disclosed malignancy, and pathology diagnosis described a gastrointestinal stromal tumor (GIST). Recurrence was diagnosed in the lungs and liver 3 years later.

GIST

GISTs are the most common mesenchymal tumors of the GI tract, with 40-70% occurring in the stomach. The annual incidence of GIST (10 to 20 cases per million) is probably underestimated, because most patients with GIST are asymptomatic until late. Ten to 30% are discovered incidentally³⁴ This is illustrated by Sanchez et al,³⁵ who reported a 0.8% incidence of GIST discovered incidentally during laparoscopic RYGBP upon inspection of the stomach before transection. All GISTs are currently considered to have some malignant potential.³⁶ In view of their degenerative potential, the difficulty to define the risk of a specific lesion without histology, and the complexity of follow-up, it is recommended to remove any such lesion discovered incidentally during a bariatric operation.

Esophageal Cancer after Bariatric Surgery

A recent meta-analysis provides evidence for an association between BMI and esophageal adenocarcinoma.³⁷ El-Serag et al³⁸ suggested that abdominal visceral adiposity might mediate most of the risk, showing a significant association with Barrett's esophagus in a model that adjusted for BMI. However, the mechanism by which this increased risk is mediated is not identified. Gastroesophageal reflux disease (GERD)^{39,40} and other factors such as hormonal, insulin, and dietetic have been proposed.⁴¹

A high incidence of GERD has also been reported after bariatric surgery, with the risk to translate into a high incidence of Barrett's metaplasia in the midterm and esophageal carcinoma in the long-term. Gastric partitioning has the potential to increase the contact period between alimentary carcinogens, the lower esophagus, and the gastric pouch.

Malabsorptive operations including the use of a long Roux-en-Y loop appear to be associated with a lower incidence of reflux symptoms.⁴² Furthermore, if we accept, as suggested by several investigators,^{43,44} that the amount of acid secretion is pivotal for development of Barrett's esophagus, the risk of esophageal cancer could even be lower after this type of bariatric operation. This is supported by the study of Csendes et al⁴⁵ who not only reported the suppression of GERD symptoms but also the regression of Barrett's metaplasia after RYGBR

The effect of gastric restrictive procedures on gastroesophageal reflux is less clear. Studies report decreased, unchanged or increased acid exposure of the esophagus after surgery,⁴⁶⁻⁴⁸ but the incidence of esophagitis appears high in most reports. This variable effect on reflux probably reflects the influence of many factors. There are differences in the way a specific procedure is carried out by different surgeons (size of pouch, position of band). GERD will also depend on the alimentary compliance, reflecting the patient's capacity to follow the dietetic guidelines and also the education and surveillance by the team in charge of the follow-up.

Four cases of adenocarcinoma of the gastroesophageal junction have been reported. One case was diagnosed 8 years after adjustable gastric banding with liver metastases.⁴⁹ Two cases were diagnosed 14 and 21 years after RYGBP,⁵⁰ pathologic study evidenced a T1N0 lesion in the first RYGBP patient, while the second patient presented with an advanced cancer involving in the entire thoracic esophagus that recurred 8 months after resection. This latter patient had been diagnosed with dysplasia 26 months before cancer diagnosis but had failed to comply with the recommended follow-up. The fourth case of adenocarcinoma of the gastroesophageal junction extended to the lesser curvature of the stomach in a 57-year-old lady after two previous bariatric operations.⁵⁰ Twenty-six years earlier, she had undergone a jejunoileal bypass which was reversed 8 years later for metabolic complications, followed by a VBG 16 years before cancer diagnosis.

Discussion

Despite the risk factors in the bariatric surgery population, few cases of cancer have been reported compared to the very large number of bariatric operations performed worldwide. Bariatric surgery may have decreased a number of risk factors for cancer, starting with elevated BMI. The time interval after surgery may, however, be too short to unveil an increased incidence of neoplasia. The low rate of cancer observed at present may also reflect the screening effect of preoperative evaluation. Prevention with careful preoperative evaluation appears to have a major role. It should start with a history for familial or patient conditions associated with a higher risk for cancer. Preoperative endoscopy is valuable to detect premalignant lesions such as metaplasia in the esophagus and stomach, for which adequate treatment or follow-up should be addressed. Presence of intestinal metaplasia in the stomach or early antral carcinoma in the massively obese individual should lead to strong consideration for prophylactic high subtotal or total gastrectomy performed using oncologic principles during RYGBP.⁵¹⁻⁵³ The importance of preoperative endoscopy is supported by Sharaf et al⁵⁴ who found that 90% of their 195 patients had one or more lesions, with 60% that changed or delayed the clinical approach, including 3% Barrett's esophagus. Similarly, preoperative endoscopic findings reported by Zeni et al⁵⁵ from 169 patients, included in addition to two cases of Barrett's esophagus, one case of GIST and one case of gastric polyp that both necessitated a partial gastrectomy. The yield is higher if the endoscopy is symptom-oriented. Endoscopy before revisional surgery for symptomatic reflux after a gastric restrictive procedure found an incidence of Barrett's metaplasia in 28% of the patients.⁵⁶ Although the influence of HP on the development of metaplasia and cancer after RYGBP is largely unknown, its demonstrated role in gastric cancer in the general population⁵⁷ justifies its treatment if discovered during preoperative evaluation.

Based on the few reported cases of cancer after bariatric surgery, evidence is lacking for the institution of a surveillance program started immediately after surgery. Continued careful reporting of complications is mandatory to delineate the risk associated with each type of bariatric operation, that could then be used to develop a surveillance algorithm for the specific populations. At present, candidates for postoperative endoscopic surveillance could include patients with a >15 year history after gastric surgery, but also patients symptomatic for GERD in whom a high incidence of Barrett's metaplasia has been reported. Routine preoperative evaluation and prompt postoperative investigation for modified or new symptoms presented by patients should lead to an individualized monitoring program in this growing population.

Published in: Obesity Surgery (2006), vol. 16, iss. 12, pp. 1656-1661. Status: Postprint (Author's Version)

References

1. Lukanova A, Bjor O, Kaaks R et al. Body mass index and cancer: results from the Northern Sweden Health and Disease Cohort. Int J Cancer 2006; 118: 458-6.

2. Bergstrom A, Pisani P, Tenet V et al. Overweight as an avoidable cause of cancer in Europe. Int J Cancer 2001; 91: 421-30.

3. Calle EE, Rodriguez C, Walker-Thurmond K et al. Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults. N Engl J Med 2003; 348: 1625-38.

4. Safatle-Ribeiro AV, Ribeiro U Jr, Reynolds JC. Gastric stump cancer: what is the risk? Dig Dis 1998; 16: 159-68.

5. Tersmette AC, Goodman SN, Offerhaus GJ et al. Multivariate analysis of the risk of stomach cancer after ulcer surgery in an Amsterdam cohort of postgastrectomy patients. Am J Epidemiol 1991; 134: 14-21.

6. Tokudome S, Kono S, Ikeda M et al. A prospective study on primary gastric stump cancer following partial gastrectomy for benign gastroduodenal diseases. Cancer Res 1984; 44: 2208-12.

7. Greene FL. Management of gastric remnant carcinoma based on the results of a 15-year endoscopic screening pro-gram. Ann Surg 1996; 223: 701-6.

8. Huang WH, Wang HH, Wu WW et al. Helicobacter pylori infection in patients with ulcer recurrence after partial gastrectomy. Hepatogastroenterology 2004; 51:1551-3.

9. Giuliani A, Caporale A, Demoro M et al. Gastric cancer precursor lesions and Helicobacter pylori infection in patients with partial gastrectomy for peptic ulcer. World J Surg. 2005; 29: 1127-30.

10. Yamamoto N, Tokunaga M, Uemura Y et al. Epstein-Barr virus and gastric remnant cancer. Cancer 1994; 74: 805-9.

11. Ovaska JT, Ekfors TO, Luukkonen PE et al. Histological changes in the gastric stump mucosa and late clinical results after Billroth I, Billroth II and Roux-en-Y operations for peptic ulcer disease. Ann Chir Gynaecol 1988; 77: 1-5.

12. Di Vita G, Siragusa G, Scozia AS et al. A comparative study of Billroth II and Roux-Y gastrojejunostomy. The mucosa of the gastric stump. Minerva Chir 1991; 46:169-73.

13. McCarthy HB, Rucker RD Jr, Chan EK et al. Gastritis after gastric bypass surgery. Surgery 1985; 98: 68-71.

14. Babor R, Booth M. Adenocarcinoma of the gastric pouch 26 years after loop gastric bypass. Obes Surg 2006; 16: 935-8.

15. Trincado MT, del Olmo JC, Garcia Castano J et al. Gastric pouch carcinoma after gastric bypass for morbid obesity. Obes Surg 2005; 15: 1215-7.

16. Sinar DR, Flickinger EG, Park HK et al. Retrograde endoscopy of the bypassed stomach segment after gastric bypass surgery: unexpected lesions. South Med J 1985; 78: 255-8.

17. Park HK, Sinar DR, Sloss RR et al. Histologic and endoscopic studies before and after gastric bypass surgery. Arch Pathol Lab Med 1986; 110: 1164-7.

18. Corsini DA, Simoneti CAM, Moreira G et al. Cancer in the excluded stomach 4 years after gastric bypass. Obes Surg 2006; 16: 932-4.

19. Escalona A, Guzman S, Ibanez L et al. Gastric cancer after Roux-en-Y gastric bypass. Obes Surg 2005; 15: 423-7.

20. Lord RV, Edwards PD, Coleman MJ. Gastric cancer in the bypassed segment after operation for morbid obesity. Aust N Z J Surg 1997; 67: 580-2.

21. Khitin L, Roses RE, Birkett DH. Cancer in the gastric remnant after gastric bypass: a case report. Curr Surg 2003; 60: 521-3.

22. Raijman I, Strother SV, Donegan WL. Gastric cancer after gastric bypass for obesity. Case report. J Clin Gastroenterol 1991; 13: 191-4.

23. De Roover A, Detry O, de Levai L. Report of two cases of gastric cancer after bariatric surgery: lymphoma of the bypassed stomach and gastrointestinal stromal tumor (GIST) after vertical banded gastroplasty. Obes Surg 2006; 16: 928-31.

24. Ruskoné-Fourmestraux A. MALT gastric lymphomas. Rev Med Interne 2004; 25:573-81.

25. Di Cosimo S, Ferretti G, Partenzi A et al. Gastric stump lymphoma five years after distal gastrectomy. Leuk Lymphoma 2003; 44: 365-7.

26. Oshita H, Tanemura H, Kanno A et al. Malignant lymphoma occurring in the residual stomach following gastrectomy: plus discussion

Published in: Obesity Surgery (2006), vol. 16, iss. 12, pp. 1656-1661. Status: Postprint (Author's Version)

based on the literature in Japan. Gastric Cancer 2003; 6: 60-3.

27. Wotherspoon AC, Ortiz-Hidalgo C, Falzon MR et al. Helicobacter pylori associated gastritis and primary B-cell gastric lymphoma. Lancet 1991; 338: 1175-6.

28. Isaacson PG. Update on MALT lymphomas. Best Pract Res Clin Haematol 2005; 18: 57-68.

29. Jain PK, Ray B, Royston CM. Carcinoma in the gastric pouch years after vertical banded gastroplasty. Obes Surg 2003; 13: 136-7.

30. Zirak C, Lemaitre J, Lebrun E et al. Adenocarcinoma of the pouch after silastic ring vertical gastroplasty. Obes Surg 2002; 12: 693-4.

31. Sweet WA. Linitis plastica presenting as pouch outlet stenosis 13 years after vertical banded gastroplasty. Obes Surg 1996; 6: 66-70.

32. Papakonstantinou A, Moustafellos P, Terzis I et al. Gastric cancer occurring after vertical banded gastroplasty. Obes Surg. 2002; 12: 118-20.

33. Hackert T, Dietz M, Tjaden C et al. Band erosion with gastric cancer. Obes Surg 2004; 14: 559-61.

34. Saund MS, Demetri GD, Ashley SW. Gastrointestinal stromal tumors (GISTs). Curr Opin Gastroenterol 2004; 20: 89-94.

35. Sanchez BR, Morton JM, Curet MJ et al. Incidental finding of gastrointestinal stromal tumors (GISTs) during laparoscopic gastric bypass. Obes Surg 2005; 15: 1384-8.

36. Saund MS, Demetri GD, Ashley SW. Gastrointestinal stromal tumors (GISTs). Curr Opin Gastroenterol 2004; 20: 89-94.

37. Hampel H, Abraham NS, El-Serag HB. Meta-analysis: obesity and the risk for gastroesophageal reflux disease and its complications. Ann Intern Med 2005; 143: 199-211.

38. El-Serag HB, Kvapil P, Hacken-Bitar J et al. Abdominal obesity and the risk of Barrett's esophagus. Am J Gastroenterol 2005; 100: 2151-6.

39. Shaheen N, Ransohoff DF. Gastroesophageal reflux, Barrett Esophagus, and Esophageal cancer. JAMA 2002; 287: 1972-81.

40. Hampel H, Abraham NS, El-Serag HB. Meta-analysis: obesity and the risk for gastroesophageal reflux disease and its complications. Ann Intern Med 2005; 143: 199-211.

41. Lagergren J, Bergstrom R, Nyren O. Association between body mass and adenocarcinoma of the esophagus and gastric cardia. Ann Intern Med 1999; 130: 883-90.

42. Ortega J, Escudero MD, Mora F et al. Outcome of esophageal function and 24-hour esophageal pH monitoring after vertical banded gastroplasty and Roux-en-Y gastric bypass. Obes Surg 2004; 14: 1086-94

43. Avidan B, Sonnenberg A, Schnell TG et al. Gastric surgery is not a risk for Barrett's esophagus or esophageal adenocarcinoma. Gastroenterology 2001; 121: 1281-5.

44. Parrilla P, Liron R, Martinez de Haro LF et al. Gastric surgery does not increase the risk of developing Barrett's esophagus. Am J Gastroenterol 1997; 92: 960-3.

45. Csendes A, Burgos AM, Smok G et al. Effect of gastric bypass on Barrett's esophagus and intestinal metaplasia of the cardia in patients With morbid obesity. J Gastrointest Surg 2006; 10: 259-64.

46. Suter M, Dorta G, Giusti V et al. Gastric banding interferes with esophageal motility and gastroesophageal reflux. Arch Surg 2005; 140: 639-43.

47. Ovrebo KK, Hatlebakk JG, Viste A et al. Gastroesophageal reflux in morbidly obese patients treated with gastric banding or vertical banded gastroplasty. Ann Surg 1998; 228: 51-8.

48. Lundell L, Ruth M, Olbe L. Vertical banded gastroplasty or gastric banding for morbid obesity: effects on gastro-oesophageal reflux. Eur J Surg 1997; 163: 525-31

49. Snook KL, Ritchie JD. Carcinoma of esophagus after adjustable gastric banding. Obes Surg 2003; 13: 800-2.

50. Allen JW, Leeman MF, Richardson JD. Esophageal carcinoma following bariatric procedures. JSLS 2004; 8: 372-5.

51. Voellinger DC, Inabnet W. Laparoscopic Roux-en-Y gastric bypass with remnant gastrectomy for focal intestinal metaplasia of the gastric antrum. Obes Surg 2002; 12: 695-8.

52. Madan AK, Lanier BJ, Tichansky DS et al. Laparoscopic Roux-en-Y gastric bypass with subtotal gastrectomy. Obes Surg 2005; 15:

1332-5.

53. Seva-Pereira G, Tombeta VL. Early gastric cancer found at preoperative assessment for bariatric surgery. Obes Surg 2006; 16: 1109-11.

54. Sharaf RN, Weinshel EH, Bini EJ et al. Endoscopy plays an important preoperative role in bariatric surgery. Obes Surg 2004; 14: 1367-72.

55. Zeni TM, Frantzides CT, Mahr C et al. Value of preoperative upper endoscopy in patients undergoing laparoscopic gastric bypass. Obes Surg 2006; 16: 142-6.

56. Balsiger BM, Murr MM, Mai J et al. Gastroesophageal reflux after intact vertical banded gastroplasty: correction by conversion to Rouxen-Y gastric bypass. J Gastrointest Surg 2000; 4: 276-81.

57. Malfertheiner P, Sipponen P, Naumann M, et al. Helicobacter pylori eradication has the potential to prevent gastric cancer: A state of the art critique. Am J Gastroenterol 2005; 100: 2100-115.