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Gastric carcinoid tumor after laparoscopic sleeve gastrectomy

Tolga Erim, D.O.[a], Yasar Colak, M.D.[a,b,*], Samuel Szomstein, M.D.[c]

[a]*Department of Gastroenterology and Hepatology, Cleveland Clinic Florida, Weston, Florida*
[b]*Department of Gastroenterology, Istanbul Medeniyet Universty, School of Medicine, Istanbul, Turkey*
[c]*The Bariatric and Metabolic Institute, Section of Minimally Invasive Surgery, Department of General Surgery, Cleveland Clinic Florida, Weston, Florida*

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Obesity is a major health and economic problem, and its prevalence has increased dramatically throughout the world [1]. This has led to an increase in bariatric treatment modalities, which results in weight loss by restriction of food intake, as well as some poorly understood neurohumoral changes in the gut–brain axis.

Carcinoid tumor is a rare neoplasm, but it is the most common neuroendocrine tumor of the stomach. Recent observations have shown a higher incidence in obese population in comparison with general population. It has also been noted that there is an increased incidence of the esophagogastric cancer after bariatric surgery procedures [2]. However, gastric carcinoid tumor (GCT) after laparoscopic sleeve gastrectomy (LSG) has not been reported so far. We report a case of GCT developing 4 years after LSG.

Case report

A 52-year-old woman was evaluated in our clinic for dyspepsia symptoms. Her physical examination was unremarkable except for obesity. Her past medical and surgical history included diabetes mellitus type 2 for 15 years, hyperlipidemia, hypertension, cholecystectomy, and LSG performed 4 years earlier for a morbid obesity, and the preoperative upper endoscopic evaluation was completely normal. Upper endoscopy revealed changes consistent with LSG and an approximately 12 mm umbilicated broad-based sessile lesion in the lesser curvature of the cardiac region of the stomach (Fig. 1B). A cold forceps biopsy had findings consistent with a well-differentiated carcinoid tumor: nesting glandular masses of monotonous small round cells with peripheral palisading, keratin and synaptophysin were positive, common leukocyte antigen was negative, Ki-67 index $<2\%$. Subsequent endosonographic evaluation performed to determine if the lesion would be amenable for endoscopic resection revealed a 10 mm x 6 mm well-demarcated, homogenous, hypoechoic lesion confined to the first and second sonographic layers (Fig. 1B). Further laboratory analysis revealed elevated gastrin level at 1291 (normal range: 0–155) pg/mL and normal chromogranin A at 3 (normal range: 0–5) mmol/L. Her colonoscopic evaluation, computed tomography scan of abdomen/pelvis, and octreotid scan were unremarkable. The tumor was resected successfully en-bloc with clear margins by endoscopic submucosal dissection (Fig. 1C). The histopathological evaluation revealed a well-differentiated GCT with clear margins of resection, and endoscopic follow-up was recommended.

Discussion

Gastric carcinoid tumor is a rare neoplasm and accounts for $<1\%$ of all gastric tumors. GCTs are subclassified as 3 distinct types based on underlying etiology and pathophysiology. Type 1 comprises 70–80% of GCTs and is closely linked to pernicious anemia and chronic atrophic gastritis. This type is associated with parietal cell loss, which leads to chronic achlorhydria, and secondary hypergastrinemia. Type 2, which accounts for 5–10%, is associated with Zollinger-Ellison Syndrome and multiple endocrine neoplasia type 1. Type 1 and 2 are usually small, multiple, and located in mucosa and submucosa and are also associated

*Correspondence: Yasar Colak, Department of Gastroenterology and Hepatology, 2950 Cleveland Clinic Boulevard, Weston, FL 33331, USA.
E-mail: dryasarcolak@yahoo.com

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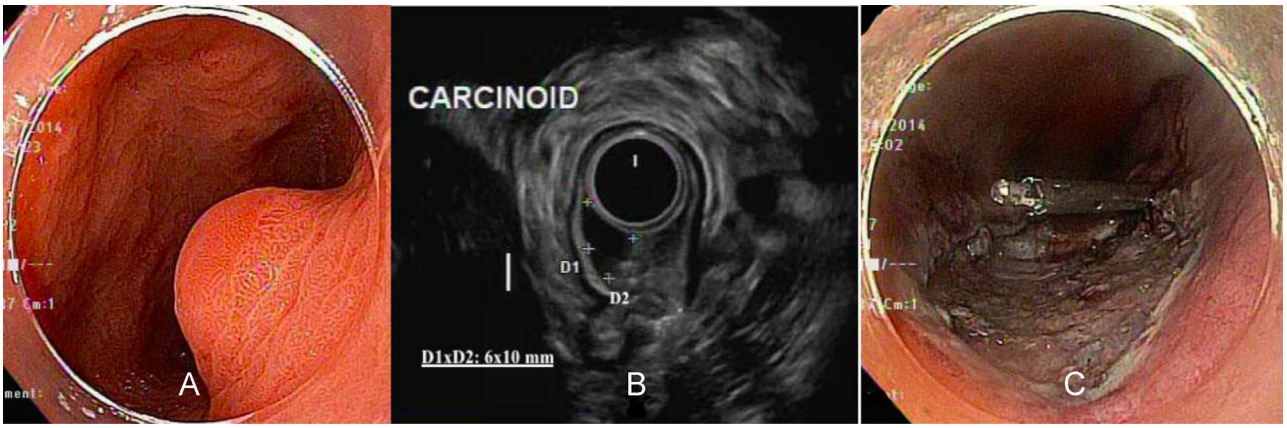

Fig. 1. The endoscopic appearance of carcinoid tumor developed after (A) laparoscopic sleeve gastrectomy and (B) the radial endosonographic appearance of tumor; (C) the endoscopic appearance of lesion area after endoscopic submucosal dissection.

with hyper gastrinemia and carry lower risk of lymph node metastasis. Type 3, which constitutes 15–25% of GCTs, is sporadic in nature and is associated with normal gastrin levels. This type is commonly single, larger, and is characterized by a more aggressive course [3].

Endoscopically, GCTs usually appear as round, nodular, or polypoid lesions. On endoscopic ultrasound (EUS) examination, they are typically hypoechoic, and homogeneous lesions are commonly located within the first and second layers (superficial and muscularis mucosa) but may invade deeper into the third layer (submucosa) and beyond (muscularis propria). Carcinoid tumors can be malignant, and this risk is generally associated with lesion size and depth of invasion. EUS is an essential diagnostic tool not only for determination of malignancy potential but also for differentiating between other lesions such as stromal tumors (i.e. gastrointestinal stromal tumors, leiomyoma). Stromal tumors commonly present as incidental findings on endoscopic. These tumors are found within the muscularis propria, occasionally within the muscularis mucosa. They usually have normal overlying mucosa but can be associated with central depression or ulceration in endoscopic evaluation. Endoscopically, GCTs may also appear very similar with the classic central umbilication/depression. Given these similarities, histologic examination and EUS evaluation are very helpful in differentiating these tumors.

An increasing incidence of GCT has been reported in large series of patients undergoing prebariatric surgery upper gastrointestinal endoscopic evaluations [4–6]. Two possible reasons were suggested by the authors: obesity-associated hormonal changes in the gut–brain axis, and further implementation of the endoscopic procedures for the purpose of preoperative control in these patient groups. However, the causal relationship is yet to be fully explained. Occurrence of gastric carcinoid tumor after sleeve gastrectomy (SG) has not previously been reported in the literature. Whether this operation has a potential role for pathogenesis of carcinoid tumors is also not known. Recent studies have shown SG-related hormonal changes in the gut–brain axis and have suggested that this may be a contributing factor to weight loss [7]. SG has been shown to lead to hypergastrinemia in rats with type-II diabetes mellitus [8]. This is similar to the physiologic changes that take place and ultimately lead to type-I GCT formation, as in our case. Our results support the hypothesis that increased gastrin levels after bariatric surgery can play role genesis of GCT. The full consequences of the changes, which take place in the gut–brain axis after bariatric surgery, are yet to be fully elucidated.

Currently, there is no consensus on endoscopic follow-up after SG. Further research is required on bariatric surgery's effect on the gut–brain-hormone axis and its possible proneoplastic consequences.

Disclosures

The authors have no commercial associations that might be a conflict of interest in relation to this article.

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