Successful Endoscopic Submucosal Dissection of Gastric Carcinoid in a Patient with Autoimmune Gastritis and Systemic Lupus Erythematosus

Tadayuki Oshima, Takuya Okugawa, Kazutoshi Hori, Yongmin Kim, Junji Tanaka, Jiro Watari and Hiroto Miwa

Abstract

A 41-year-old woman was referred with epigastric discomfort. She had a 20-year history of SLE. Esophagastroduodenoscopy (EGD) examination showed severe atrophic gastritis in the stomach and a protruding lesion was detected. Histological examination showed a carcinoid tumor with cytoplasmic staining with chromogranin-A. Using an endoscopic submucosal dissection (ESD) technique, en bloc resection of the tumor was performed. No recurrence has been found for 3 years after the treatment. Type I gastric carcinoid can occur at an earlier age with AIG and autoimmune diseases such as systemic lupus erythematosus (SLE). This is the first report of gastric carcinoid that was treated by ESD.

Key words: carcinoid, SLE, autoimmune gastritis, ESD

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Introduction

Type I gastric carcinoids are associated with autoimmune atrophic gastritis (AIG) and endocrine hyperplasia, commonly in combination with pernicious anemia arising from vitamin B12 deficiency (1). Associations of carcinoids with autoimmune disorders other than AIG have rarely been reported (2). A rare case of gastric carcinoid in a relatively young woman with AIG and systemic lupus erythematosus (SLE) treated by endoscopic submucosal dissection (ESD) is reported.

Case Report

A 41-year-old woman was referred to our department with epigastric discomfort. She had a 20-year history of SLE and was taking 20 mg/day of corticosteroid. Esophagastroduodenoscopy (EGD) examination showed severe atrophic gastritis in the body of the stomach (Fig. 1A) and no atrophy in the antrum (Fig. 1B). A protruding lesion was detected at the anterior wall of the body (Fig. 1A and B). Endoscopic ultrasonography (EUS) showed a protruding 5-mm diameter lesion in the mucosal layer not affecting the submucosal layer (Fig. 1D). Histological examination showed a carcinoid tumor with cytoplasmic staining with chromogranin-A. Atrophic gastritis, intestinal metaplasia, and endocrine cell hyperplasia were identified (Fig. 2A and B). Micronests of endocrine cells were also detected at another part of the gastric body (Fig. 2C). Diagnosis of the co-existence of AIG was supported by the detection of both anti-parietal cell antibody and anti-intrinsic antibody and an elevated blood gastrin level (3,400 pg/mL). Anti-*Helicobacter pylori* IgG was negative. Laboratory examinations demonstrated no anemia. Anti-nuclear antibody and anti-ds DNA antibody were positive at the time of the diagnosis of SLE.

Using ESD technique, en bloc resection of the tumor was performed, and the residual tumor was completely resected histologically (Fig. 3). Oral administration of steroid was continued before and after the operation and no complication occurred. Histological examination of the resected specimen revealed carcinoid tumor with slight submucosal invasion (sm1) without lymph or vascular invasion. The le-
sion was strongly positive for synaptophysin and chromogranin-A. Less than 1% of cells were Ki-67-positive. Endoscopic surveillance has been performed every 6 months because type I carcinoid can recur in other parts of the gastric body. No recurrence has been found for 3 years after the treatment. However, continuous surveillance is needed.

Discussion

In AIG, chronic exposure to elevated serum gastrin due to gastric atrophy affects the development of gastric carcinoid. Therefore, the average age at detection of gastric carcinoids is in the 60s (3), and it rarely occurs at younger ages. In cases with other autoimmune disorders (4, 5), type I gastric carcinoids may develop at a relatively young age. Pernicious anemia is usually already present when type I gastric carcinoid is detected. This may be related to the opportunity of examining the stomach in a patient with anemia. These lesions were reported to be detected in up to 9% of patients with pernicious anemia (6). It may be difficult to find these lesions without clinical manifestations of anemia, and consequently it is difficult to know the exact incidence of gastric carcinoid in AIG. Furthermore, the prevalence and the association of SLE and AIG itself are still unclear. The association of SLE and type I gastric carcinoid has rarely been reported previously (5, 7, 8). Although all three previously reported cases with type I gastric carcinoid with AIG and SLE showed pernicious anemia at the time of finding carcinoids (5, 7, 8), in the present case, vitamin B12 was low but pernicious anemia was not present when the carcinoid was detected, indicating that gastric carcinoid possibly occurred before the induction of pernicious anemia. These data also indicate that AIG developed late after the diagnosis of SLE, and autoimmune disorders might have accelerated the appearance of gastric carcinoid at an earlier age without pernicious anemia. Further cases are needed to elucidate the mechanisms of the early development of type I gastric carcinoid with autoimmune diseases other than AIG.

Antrectomy has been performed for type I carcinoid tumors to remove the cells that produce gastrin. However, Gilligan et al. (9) suggested endoscopic treatment for type I gastric carcinoids of less than 1 cm in diameter and fewer than 3-5 in number. The recent development of ESD technique enables the complete resection of these lesions. Although endoscopic mucosal resection (EMR) was first introduced for the treatment of type I gastric carcinoid (10), ESD is a better technique than EMR to resect tumors like carcinoid, which often invade the submucosal layers, because the resecting plane can be easily checked while cutting. The depth of invasion and the lymphovascular invasion of the resected specimens can be precisely assessed by en bloc resections. Although the depth of the lesion in this case was to the submucosal layer (sm1), after careful discussion with the patient, she chose not to receive additional treatment including surgery. She has been followed-up for 3 years without needing any additional
In summary, the successful treatment of type I gastric carcinoid by ESD in a patient with SLE was presented. This case report highlights the fact that gastric carcinoid can occur at an earlier age in patients with AIG and autoimmune diseases such as SLE. This is the first reported case of gastric carcinoid that was treated by ESD.

The authors state that they have no Conflict of Interest (COI).

References


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