Successful type-oriented endoscopic resection for gastric carcinoid tumors: A case report

Shouji Shimoyama, Mitsuhiro Fujishiro, Yutaka Takazawa

Abstract

The standard treatment in Japan for gastric carcinoid has been gastrectomy with lymphadenectomy. This report describes the possibility of endoscopic treatment as an appropriate option for gastric carcinoid fulfilling certain conditions. A 46 year old woman underwent endoscopic mucosal resection for two 3 mm gastric carcinoids. The patient had hypergastrinemia with pernicious anemia and type A chronic atrophic gastritis, suggesting that the tumors were type I in Rindi’s classification. Both tumors were located in the mucosal layer with no cellular polymorphism and were chromogranin A positive. Neither tumor recurrence in the stomach nor distant metastases have been documented during the 5 years of follow-up. Although many type I gastric carcinoids may be clinically indolent, reports on successful endoscopic treatment for this carcinoid have been scanty in the literature in Japan, presumably because of the hitherto surgical treatment stance for the disease. This report discusses how the size, number, depth and histological grading of the type I gastric carcinoid could allow the correct identification of a benign or malignant propensity of an individual tumor and how endoscopic resection could be a treatment of choice when these factors render it feasible. This stance could also obviate unnecessary surgical resection for more benign tumors.

Key words: Endoscopic resection; Gastric carcinoid; Hypergastrinemia; Pernicious anemia; Type A chronic atrophic gastritis.

INTRODUCTION

Gastric carcinoids (GCDs) were previously thought to be extremely rare in the West, constituting only 2.6% of all gastrointestinal carcinoids in the 1950s[1]. Their incidence, however, has chronologically increased to 8.7% in the 1990s[2]. Interestingly, GCDs, the second most common (21%-27%) gastrointestinal carcinoids in Japan[3], have also seen an increase in cases over the past 5 decades[4]. These trends may be due to an actual increase but the more likely reason is improvements in diagnostic technology and increased awareness. Despite the steady rise in the incidence of GCDs in the gastrointestinal tract in both regions, GCDs have been considered to be a curiosity accounting for less than 1%[5] of all gastric tumors and such rarity has made it difficult to understand precisely
the biological nature of them and to establish the optimal treatment options for the disease.

GCDs are an enigmatic malignancy that, while slow in growth compared with adenocarcinoma, can sometimes behave aggressively. This has led to a debate concerning the optimal treatment for GCDs. In Japan, radical gastrectomy has been recommended as a general treatment for them due to the concern over the substantial metastatic rates (4.6%-30%) even among small and/or submucosal GCDs\(^3,4,6,7\). On the other hand, Western researchers have recently proposed a spectrum of treatment options for GCDs\(^8\) ranging from less invasive endoscopic polypectomy to more aggressive surgery on the basis of the background gastric pathological characteristics with or without hypergastrinemia as a pathogenetic trait\(^9-11\).

Here we report a case of GCDs with hypergastrinemia successfully treated by endoscopic mucosal resection (EMR) followed by no evidence of recurrence for 5 years. Because of the hitherto aggressive treatment stance in Japan, cases of successful endoscopic treatment for GCDs have been scarce in the literature. This report raises the possibility that pathobiological analyses of individual GCDs could select patients to benefit from less invasive treatment, so realizing type-oriented patient management.

**CASE REPORT**

A 46 year old woman underwent upper gastrointestinal endoscopy in 2003 due to upper abdominal discomfort. Endoscopic examination revealed two tiny elevated lesions 3 mm in diameter located on the anterior and posterior walls of the upper third of the stomach (Figure 1). Atrophy was more marked in the body-fundus than in the antrum. Biopsy specimens from both lesions showed microlobular-trabecular cell clusters with no cellular polymorphism. No extragastric hormonal syndromes such as flushes or diarrhea were identified. Patient interview revealed a previous diagnosis of pernicious anemia at the age of 30 and investigation showed combined iron (56 mg/dL) and vitamin B\(_12\) (230 pg/mL) deficiency anemia with low levels of hemoglobin (10.4 g/dL) and mean corpuscular volume (89.6 fL). The positivity of both anti-parietal cell and anti-intrinsic factor antibodies, as well as corpus predominant atrophic gastritis and elevated serum gastrin level (3827 pg/mL), suggested that the elevated lesions were type I\(^9-11\) carcinooid tumor associated with pernicious anemia and type A chronic atrophic gastritis (CAG/A)\(^12\). Endoscopic ultrasonography failed to evaluate the tumor depth definitively. There was no evidence of lymph node or liver metastases. She had been diagnosed with epilepsy 30 years prior to this visit and sodium valproate had been prescribed since then. Continuous prescription of proton pump inhibitors was not confirmed. After fully informed consent, she underwent cap-assisted EMR, an “inject, suck and cut” technique, for both lesions in July 2004. The postoperative course was uneventful.

Both resected specimens showed a histological architecture of microlobular-trabecular cell clusters in the mucosal layer with marked fundic gland atrophy (Figure 2). Endocrine cell micronests were observed in the mucosal layer and in the lamina propria mucosa. Neither cellular polymorphism nor mitoses were observed. Neither lymphatic nor vascular invasion were documented. Both tumors as well as endocrine cell micronests were chromogranin A positive (Figure 2, inset). All resection margins were negative for carcinoid cells.

Under the postoperative annual endoscopies, any lesions of concern for the endoscopist were biopsied and there has been no evidence of tumor recurrence in

Figure 1  Tiny elevated lesions, 3 mm in diameter detected on the posterior (A) and the anterior (B) walls of the upper third of the stomach.

Figure 2  Histological findings of the tumor located on the posterior wall of the stomach (Hematoxylin-eosin stain, × 40). The tumor exhibits microlobular-trabecular growth patterns with chromogranin A positive (inset, × 100). No cellular polymorphism is observed. The other tumor showed the same findings.
December Tumors showed prevalence of solid cellular aggregates and large trabeculae, crowding and irregular distribution of round to spindle and moderately polymorphic nuclei of larger size, often with evident nuclei and rather few, morphologically typical mitoses.

Grade 2 Tumors showed prevalence of solid cellular aggregates and large trabeculae, crowding and irregular distribution of round to spindle and polyhedric tumor cells, fairly large vesicular nuclei with prominent eosinophilic nucleoli or smaller, hyperchromatic nuclei with irregular chromatins clumps and small nucleoli, considerable mitotic activity, sometimes with atypical mitotic figures and scant necrosis.

Grade 3 Tumors showed severe histological atypia with solid to diffuse structure and frequent central necrosis. They were composed of tightly packed, small to mid-sized tumor cells showing large, irregular, polymorphic and hyperchromatic nuclei, scant cytoplasm and frequent, often atypical, mitosis.

Table 1 Histological tumor grading proposed by Rindi et al. (14)

| Grade | Description |
|-------|-------------|
| Grade 1a | Tumors characterized by small and microlobular-trabecular aggregates formed by regularly distributed, often aligned cells with regular monomorphic nuclei, usually inapparent nucleoli, rather abundant fairly eosinophilic cytoplasm and almost absent mitoses. |
| Grade 1b | Tumors characterized by significant areas with solid structure, absence of cell alignment, round to spindle cell shape, irregular and moderately polymorphic nuclei of larger size, often with evident nuclei and rather few, morphologically typical mitoses. |
| Grade 2 | Tumors showed prevalence of solid cellular aggregates and large trabeculae, crowding and irregular distribution of round to spindle and polyhedric tumor cells, fairly large vesicular nuclei with prominent eosinophilic nucleoli or smaller, hyperchromatic nuclei with irregular chromatins clumps and small nucleoli, considerable mitotic activity, sometimes with atypical mitotic figures and scant necrosis. |
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discussion

The optimal treatment options for GCDs have not been precisely defined. Earlier Japanese literature reviews or case collections elucidated that the risk of metastasis depended on the tumor size and depth. Only minute (< 0.5 cm in diameter) GCDs showed no metastases but then began to spread outside the stomach in correlation with tumor size(1), the incidences being 6.7% for < 1 cm, 27.7% for < 2 cm and 45.8% for < 3 cm in diameter(3). In addition, metastatic rates of GCDs situated in mucosal, submucosal and proper muscle layers were 7.5%, 13.2%-15.5% and 44.8% respectively(4,6). Even small submucosal GCDs (< 1.0 cm) were found to metastasize at a substantial rate (7.9%)(8) equal to or even higher than those of submucosal gastric cancer(11), suggesting that GCDs often metastasize even when they are small (< 1 cm) or confined to the submucosal layer. Therefore, in Japan, total or subtotal gastrectomy with lymphadenectomy has been recommended and indeed performed for GCDs, irrespective of size, depth or number.

On the other hand, an Italian research group(9,10) has proposed a new classification for GCDs by dividing them into three types: type I is those arising in CAG/Á with hypergastrinemia; type II occurs in patients with hypergastrinemia due to the Zollinger-Ellison syndrome in association with multiple endocrine neoplasia type 1; and type III is sporadic GCDs not associated with any specific pathogenetic background. This classification is of great worth because of its ability to predict the biological aggressiveness of GCDs. Types I and II GCDs were low grade tumor diseases with excellent prognosis although a relatively higher degree of aggressiveness was observed for type II whereas those independent of gastrin promotion (type III) were life-threatening neoplasms(9,11). Metastatic rates were 0%-7.8% in type I, 18.1%-30.0% in type II and 16.7%-75.0% in type III tumors(12-14,16,17). Type I GCDs were mainly restricted to the mucosa or submucosal layer and were usually smaller in size at presentation(9,11,16) whereas increasing type numbers (from type I to III) correlated with deeper tumor infiltration and larger tumor size. Even a conservative approach for type I GCDs was proposed by observations of spontaneous regression(15) or the absence of clinical problems(16) for varying periods of follow-up. These observations suggest that type I GCDs will not become clinically overt and that endoscopic treatment is considered safe.

Against this background, Gilligan et al.(8) advocated a treatment algorithm for GCDs, including parameters of the above-mentioned subtypes as well as sizes and numbers of the tumors. In types I and II GCDs, initial treatment is an endoscopic polypectomy for less numerous (< 3-5 lesions) and smaller (< 1 cm) tumors and antrectomy or local resection for more numerous (> 3-5 lesions) and larger (> 1 cm) ones. Both treatments should be followed by endoscopic surveillance biannually and any recurrence should be treated by local excision, antrectomy or wider gastrectomy. On the other hand, en bloc surgical resection with lymphadenectomy is recommended for type III tumors. Subsequently, the rationale for this type-oriented treatment has been confirmed by prospective(18) and retrospective(19) studies. In addition, guidelines for gastrointestinal endocrine tumors from the United Kingdom have stated that surveillance only is considered appropriate for many type I GCDs(20).

The Japanese aggressive treatment stance thus far has been based on cases of small but node-positive GCDs. Taking the tripartite classification into account, however, these tumors presumably comprise of pathobiologically heterogeneous types of neoplasms because they were not stratified by subtype in some reports(21) or were at least non-type I in others(22,23). Nevertheless, it is also a fact that type I GCDs may occasionally countermand the anticipated biological behavior(14,16,24). In this regard, histological grading (Table 1) and tumor depth(14,16,24) have been demonstrated to be characteristics by which individual tumor aggressiveness is predictable with a higher accuracy than would be by simple tripartite classification. Therefore, integration of these factors into the Gilligan’s decision tree could allow more correct identification of benign or malignant propensities in individual tumors and endoscopic treatments such as EMR and endoscopic submucosal dissection (ESD) could be a treatment of choice when size, number, depth and histological grading of a tumor render them feasible. These stances

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are in accordance with those published very recently\(^2\) and can help avoid any unnecessary gastrectomy for type I GCDs with the more benign phenotype\(^2\), something which undoubtedly impairs personal well-being without any advantage.

The selection of endoscopic treatment modalities depends on the size and degree of the submucosal involvement of the target lesion. In general, EMR is applied for smaller (e.g. < 1 cm) lesions without submucosal invasion or fibrosis\(^2\) whereas ESD, an “inject, incise the mucosa and dissect the submucosa” technique, is applied for lesions larger in size and/or with some submucosal involvement\(^2\). The goal of both techniques is an en bloc resection realizing a precise histological diagnosis. EMR, by the nature of its technique, could achieve more increased en bloc and histologically complete resection rates compared with EMD but is associated with longer average operation times and a higher incidence of intraoperative bleeding and perforation\(^2\). In this case, we consider that intramuscular and small (3 mm each) lesions render EMR feasible.

Even after Gilligan’s proposal and in the era of technically advanced endoscopic resection, reports in Japan on GCDs associated with hypergastrinemia with a successful resultant of endoscopic treatment or follow-up only have remained rare in the literature, probably due to the less common consideration of the GCD classification (Table 2)\(^2\). In the present case, the Gilligan’s recommendation and the intramuscular localization with a histologically less aggressive grade of tumor justify the endoscopic resection and repeated follow up endoscopies as a treatment strategy. Despite conditions of persistent hypergastrinemia, a relatively longer tumor free period of 5 years as compared with those (between 9 mo and 12 years) in the reported cases in the literature confirms the rationale of our strategy.

Pernicious anemia or CAG/A predispose the development of both gastric cancer and GCDs\(^1\) as separated\(^2\) or mixed\(^3,4\) tumors, underscoring the importance of continuous repeated endoscopic monitoring for type I GCDs even after successful endoscopic resection. One of the presumed underlying mechanisms is a trophic effect and tumorigenic potential of inappropriately sustained hypergastrinemia. Awareness of these facts is important at each step of the sequence of patient management, i.e. at the time of diagnosis, treatment and each follow-up examination.

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