

Synchronous Occurrence of Early Gastric Cancer and a Gastrointestinal Stromal Tumor at the Same Site: A Case Report

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Abstract

A 70-year-old male was admitted to our institution with a complaint of vomiting in mid-September 2011. Upper gastroendoscopy revealed an approximately 20 mm submucosal tumor in the anterior wall of the lesser curvature at the lower gastric body that was overlain by 15 mm type 0-I conglomerate lesion. Distal gastrectomy and reconstruction using Billroth I was performed in mid-November 2011. Gross specimen revealed an 18×18×10 mm elastic, hard submucosal tumor in the lesser curvature at the lower body, which was overlain by a 14×14×8 mm type 0-I conglomerate lesion. Hematoxylin and eosin staining of the submucosal tumor revealed spindle cells, positive for c-kit and CD34, but negative for desmin and S-100 proteins after immunochemical analysis; thus, an uncommitted type of gastrointestinal tumor (GIST) was diagnosed. Pathological analysis of the type 0-I lesion revealed a well differentiated tubular adenocarcinoma. There was no continuity between gastric cancer and GIST. His postoperative course was uneventful and the patient was discharged ambulatory 25 days after admission. This case represents a rare case of synchronous occurrence of early gastric cancer and GIST at the same site.

Keywords: Early gastric cancer; Gastrointestinal stromal tumor

Introduction

Gastrointestinal stromal tumor (GIST) is a rare form of mesenchymal tumor of the gastrointestinal tract, accounting for 1% of malignant neoplasms [1]. Most gastrointestinal mesenchymal tumors are GISTs, originating from interstitial cells of Cajal or precursors of these cells. Although several reports regarding GIST have incrementally advanced our understanding of this condition in recent years, reports of synchronous occurrence of GIST and adenocarcinoma have been rare.

Case Presentation

A 70-year-old male was admitted to our institution with a complaint of vomiting and epigastric discomfort in mid-September 2011. His past history included vertigo at the age of 69, which was successfully treated using medication. He admitted to a habit of alcohol use, consuming approximately 200mL whisky each day, but denied tobacco use. His family histories excluded GIST or some digestive cancer. He had occasionally experienced epigastric discomfort. His blood pressure was 120/72 mmHg, heart rate was 75 beats/min, and body temperature was 35.4°C.

On clinical examination, his weight was 66 kg, height was 168 cm, and body mass index was 23.4 kg/m². Inspection of the palpebral conjunctiva revealed evidence of anemia. The abdomen was flat with normal peristalsis; moreover, tenderness was not observed in the upper abdomen. No palpable mass or signs of peritoneal irritation were observed over the abdomen. Blood chemistry analyses revealed the following: mild inflammation (white blood cell count, 9,900/μL and C-reactive protein levels, and 1.6 mg/dL), slightly elevated γ-glutamyl transpeptidase (56 IU/L), and slightly decreased potassium levels (3.3 mEq/L). Tumor marker levels, including serum carcinoembryonic antigen and serum carbohydrate antigen 19-9, were within normal limits. Upper gastroendoscopy revealed severe atrophic gastritis and an approximately 20 mm submucosal tumor in the anterior wall of the lesser curvature at the lower gastric body, which was overlain by a 15 mm type 0-I conglomerate lesion (Figure 1a). An endoscopic biopsy from the conglomerate lesion indicated group V. Moreover, *H. pylori* infection was also revealed. Endoscopic ultrasound revealed the

invasion of cancer cells into the submucosal layer and a hypochoic mass in the fourth layer of the gastric wall (Figure 1b). Whole body computed tomography revealed absence of metastasis including lymph nodes. Early gastric cancer (cT1bN0M0, Stage IA) with a submucosal tumor was diagnosed and surgical resection was planned.

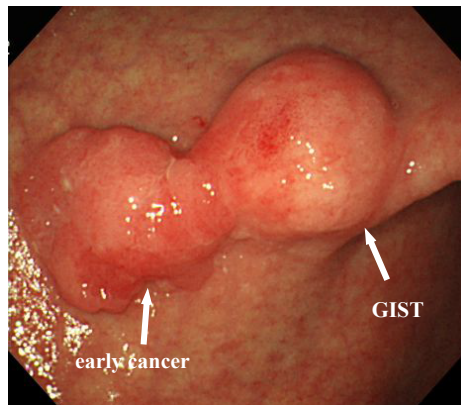
Distal gastrectomy using lymph node dissection (D1) and reconstruction using Billroth I was performed in mid-November 2011. Gross specimen revealed an 18×18×10 mm elastic hard submucosal tumor in the lesser curvature at the lower body, which was overlain by a 14×14×8 mm type 0-I conglomerate lesion (Figure 2). Hematoxylin and eosin staining of sections from the submucosal tumor revealed spindle cells with almost no mitosis or necrosis (Figure 3a) and staining of epithelial tumor revealed a well differentiated tubular adenocarcinoma (Figure 3b). There was no continuity between gastric cancer and GIST (Figure 3c). The labeling index for Ki-67, determined by counting positively stained nuclei, was approximately 1% (Figure 3d). Mitotic activity was < 5/50 HPF and tumor size was < 5%; thus, this gastric GIST was classified as none or the very low risk category according to the Miettinen or Fletcher's classifications, respectively. Immunochemical analysis of tumor cells revealed positive staining for c-kit and CD34, but no staining for desmin or S-100 proteins (Figures 3e-h); thus, an uncommitted type of GIST was diagnosed. The adenocarcinoma was invaded into submucosa with a single lymph node metastasis of No. 3 (D1). Moreover, vessel invasion was not observed, but lymphatic invasion was recognized. No cancer cells were found in the resected stump. Postoperative diagnosis was early gastric cancer of pT1bN1M0,

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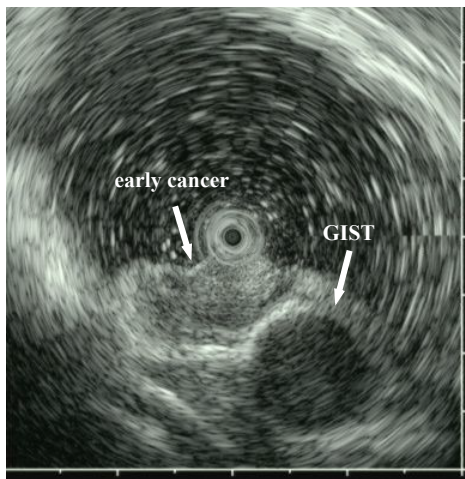
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a



b

Figure 1: Upper gastroendoscopy and endoscopic ultrasonography findings. Upper gastroendoscopy revealed an approximately 20-mm submucosal tumor in the anterior wall of the lesser curvature at the lower gastric body, which was overlain by a 15 mm type 0-I conglomerate lesion (a). Endoscopic ultrasonography revealed invasion of cancer cells into the submucosal layer and a hypoechoic mass in the fourth layer of the gastric wall (b).

Stage IB. His postoperative course was uneventful, and the patient was discharged ambulatory 25 days after admission.

Discussion

Gastric adenocarcinoma and GIST are distinct neoplasms originating from different cell layers; gastric adenocarcinomas are derived from epithelial layers and GISTs are derived from nonepithelial layers. The synchronous occurrence of gastric cancer and GIST has rarely been reported in the literature [2-10]. Close existence of gastric cancer and GIST has been described in some literature [2,3,5], whereas gastric cancer and GIST are reported to exist separately in the stomach in others [4,6-10]. As for cases in which gastric cancer and GIST closely existed [2,3,5], detailed information was reviewed. Marionara et al. [2] reported an 81-year-old female who presented 4cm well-differentiated adenocarcinoma and 5cm GIST from the fundus to the cardia, which was treated with partial gastrectomy. In the Marionara's case, the biological activity of GIST was not described. Liu et al. [3] reported a 70-year-old male who presented with 8×5×3 cm adenocarcinoma and GIST from the cardia to the upper gastric body, which was treated with

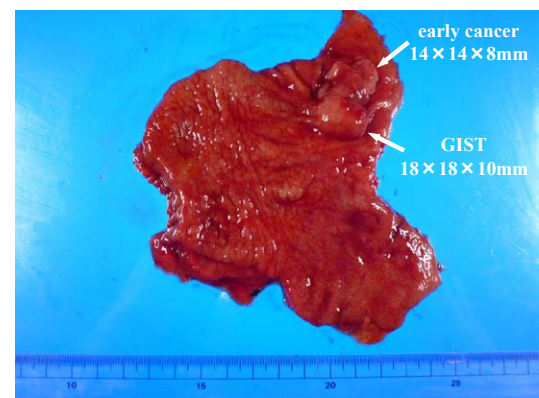
total gastrectomy. In the Liu's case, the biological activity and the size of GIST was not described. Rauf et al. [5] reported a 70 year old male who presented with 10×6×4 mm poorly differentiated adenocarcinoma and 2×2 cm of low risk GIST (Mitotic activity was < 5/50 HPF) from the gastric body to the antrum, which was treated with total gastrectomy. As there are few case reports concerning the synchronous occurrence of gastric cancer and GIST, further accumulation of reports are needed.

The reason for synchronous occurrence of GIST and gastric cancer is unknown, but plausible reasons include the following:

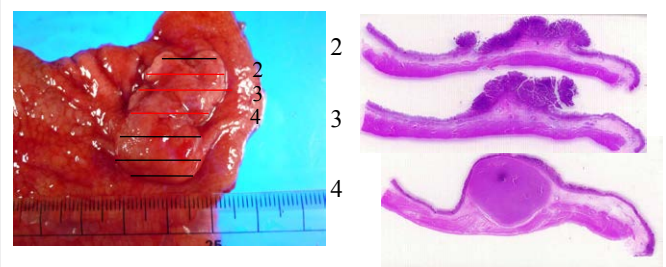
- 1) Mechanical stimulation or blood disturbance caused by GIST that affects gastric cancer,
- 2) The production of some tumor growth promoting substances by GIST, and
- 3) The mere incidental occurrence.

Maiorana et al. [2] supposed that genetic mutations or a single carcinogenic agent may interact with two neighboring tissues to induce the development of different histotypes in the same area.

However, Liu et al. [11] reported 0.74% of patients with upper gastrointestinal malignancies accompanied by GIST, although Chan et al. [12] 5.3% operated cases of non-GIST gastric neoplasm were synchronously accompanied by gastric GIST (non-GIST gastric neoplasm and gastric GIST were not necessarily located at the same time). The incident of synchronous occurrence of gastric cancer and GIST may be not uncommon, but the synchronous occurrence of early gastric cancer and GIST at the same site is comparatively rare. Although the incidence of the simultaneous occurrence of gastric cancer and GIST at the same site is unknown, further research regarding the



a



b

Figure 2: Gross specimen. Gross specimen revealed an 18×18×10 mm elastic, hard submucosal tumor in the lesser curvature at the lower body, which was overlain by a 14×14×8-mm sized type 0-I conglomerate lesion (a). There was no continuity between gastric cancer and submucosal tumor (b).

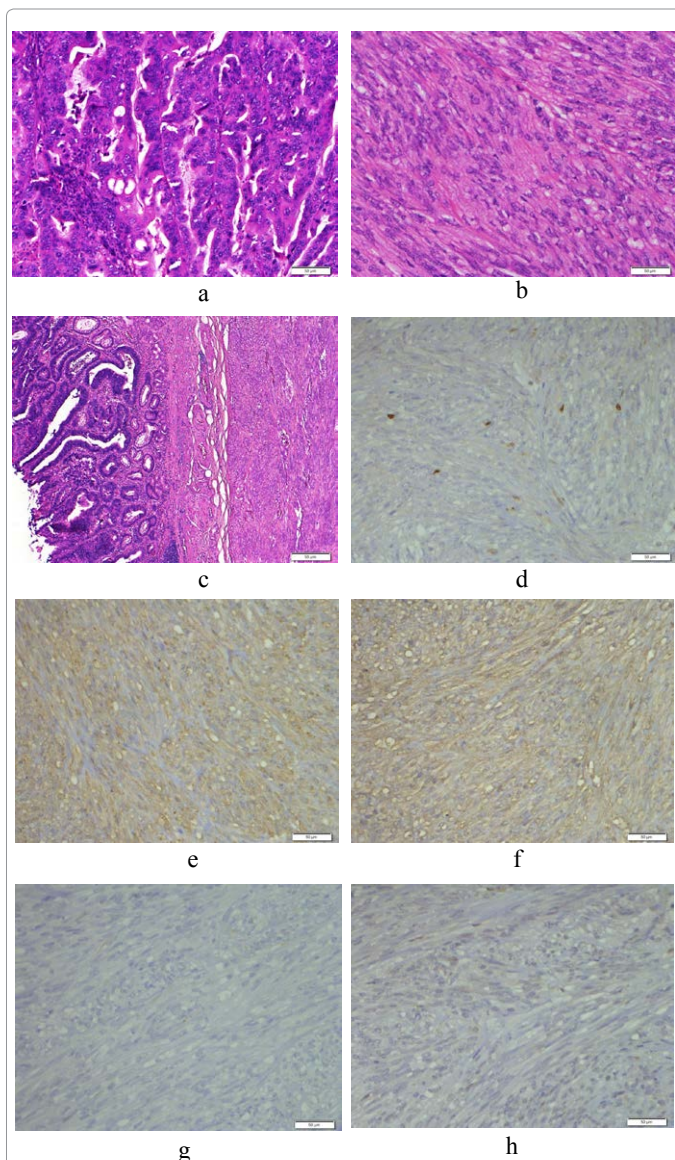


Figure 3: Histopathological findings. Hematoxylin and eosin staining of the submucosal tumor revealed spindle cells with almost no mitosis or necrosis (a). Whereas staining of the epithelial tumor revealed a well differentiated tubular adenocarcinoma (b). There was no continuity between gastric cancer and GIST (c). The labeling index for Ki-67, determined by counting positively stained nuclei, was approximately 1% (d). (a,b,d: high power field, $\times 400$, c: low power field, $\times 100$) (d). Immunohistochemical staining of tumor cells revealed positive staining for c-kit (e) and CD34 (f), but negative staining for desmin (g) and S-100 proteins (h). (e-h: high power field, $\times 400$).

underlying mechanism of simultaneous occurrence of distinct tumors is hoped.

Conclusions

Synchronous occurrence of early gastric cancer and GIST at the same site of the same organ may be a rare finding. Further investigation regarding the mechanism of synchronous occurrence of two different tumors originating from distinct cell layers, including those caused by genetic mutation and carcinogenic agents, will be needed to understand these associations.

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