Takeuchi et al., J Clin Case Rep 2014, 4:4
DOI: 10.4172/2165-7920.1000352

Journal of Clinical Case Reports

Case Report Open Access

Endoscopic Submucosal Dissection for Early Gastric Cancer Complicated by Gastric Gland Heterotopia: A Case Report

Nobuhiro Takeuchi[1]*, Yusuke Nomura[1], Yu Nishida[1], Tetsuo Maeda[1], Hidetoshi Tada[1] and Kazuyoshi Naba[2]

[1]*Department of Gastroenterology, Kawasaki Hospital, Kobe, Japan*
[2]*Department of Laboratory Medicine, Kawasaki Hospital, Kobe, Japan*

Abstract

A 72-years-old male visited our institution because of severe abdominal distention. Abdominal computed tomography revealed liver cirrhosis with massive retention of ascites. Ascites was treated using diuretic drugs and albumin preparation. Gastroendoscopy revealed esophageal varices, which was successfully treated with endoscopic variceal ligation. A follow-up gastroendoscopy performed 4 months later revealed the disappearance of esophageal varices and the presence of a depressed lesion on the anterior wall of the lesser curvature of the mid-gastric body with a small orifice near the anal side of the depressed lesion, suggesting gastric gland heterotopia. A biopsy from the depressed lesion revealed group 5. Endoscopic ultrasonography revealed anechoic lesions in the third layer and type 0-IIc lesion with invasion to the third layer, suggesting that the IIc lesion invaded the submucosal layers. On the basis of endoscopic findings, the IIc lesion was considered to be within the submucosal layer; therefore, Endoscopic Submucosal Dissection (ESD) was performed, and pathological findings of the resected specimen revealed moderately differentiated tubular adenocarcinoma within the mucosal layer and multiple cystic dilated lesions in the submucosal layer. The post-ESD course was uneventful and recurrence or de novo lesion has not been detected by regular gastroendoscopy.

Keywords: Gastric gland heterotopia; Endoscopic submucosal dissection; Abdominal distention

Introduction

Gastric Gland Heterotopia (GGH) is defined as the proliferation of ectopic gastric glands under the submucosal layer. In clinical situations, when GGH is found, it should be carefully assessed because GGH sometimes accompanies multiple gastric cancers. Here we report a case of early gastric cancer complicated by GGH, which was treated by ESD.

Case Report

A 72-year-old male visited our institution because of severe abdominal distention in February 2011. Past medical history included myocardial infarction, which was treated with a coronary stent. Abdominal computed tomography revealed retention of massive ascites and liver cirrhosis. Ascites was treated with diuretics and albumin preparation. Hepatitis B surface antigen was positive and hepatitis C antibody was negative. Therefore, he was diagnosed with hepatitis B-related cirrhosis. Gastroendoscopy revealed esophageal varices, which was successfully treated with endoscopic variceal ligation. Follow-up gastroendoscopy performed in June 2011 revealed the disappearance of esophageal varices and the presence of a depressed lesion on the anterior wall of the lesser curvature of the mid-gastric body; therefore, he was admitted to our institution for further examination and treatment. On admission, examination of the palpebral conjunctiva revealed mild anemia. Chest auscultation revealed no abnormal findings. The abdomen was soft and flat with normal bowel sounds and no palpable lymph nodes. Blood chemistry analyses revealed mild anemia (red blood cell counts, $346\times10^4/\mu L$, hemoglobin level, 10.0 g/dL), mildly increased gamma-glutamyl transpeptidase level (57 IU/L), and mildly increased carbohydrate antigen 19-9 level (55.2 U/mL) irrespective of the normal carcinogenic embryonic antigen level (4.4 ng/mL). Abdominal radiography revealed normal gas distribution. Further inspection by gastroendoscopy revealed a small orifice near the anal side of the depressed lesion (Figure 1a), which appeared to be gastric gland heterotopia (GGH). Subsequently, narrow-band imaging (NBI) endoscopy (Figure 1b and 1c) revealed irregular vascular patterns on the surface structures of the depressed lesion, which was consistent with the findings of gastric cancer. A biopsy from the depressed lesion revealed group 5. Endoscopic ultrasonography (EUS) (Figure 1d) revealed an anechoic lesion in the third layer, and the type 0-IIc lesion seemed to reach the third layer, suggesting that the type 0-IIc lesion had invaded the submucosal layer. On the basis of endoscopic findings, the type 0-IIc lesion was considered to be within the submucosal layer; therefore, this lesion was a candidate for Endoscopic Submucosal Dissection (ESD). Subsequently, ESD was performed in mid-July 2011. Pathological findings of the resected specimen (Figure 2a-2c) revealed moderately differentiated tubular adenocarcinoma within the mucosal layer and multiple cystic dilated lesions in the submucosal layer. The postoperative course was uneventful and he was discharged in late-July. Till date recurrence or de novo lesion has not been detected by regular follow-up gastroendoscopy.

Discussion

GGH was first reported by Scott et al. [1] in 1947 as diffuse congenital cystic hyperplasia; subsequently, Oberman et al. [2] reported the condition as diffuse heterotopic cystic malformation. Wagner and Tcherkoff [3] reported it as diffuse cystic malformation of the stomach. GGH predominantly affects 50-60 year old males. The frequency of GGH is reported to be 5.7%. Its favored sites of involvement are the posterior side of the mid-gastric body, and borderline of the fundic and pyloric gland regions [4]. GGH usually forms diffuse lesions. GGH can be complicated by cancers (33%) [4]; 80% of them being differentiated by tubular adenocarcinomas with invasion to the mucosal

***Corresponding author:** Nobuhiro Takeuchi, 3-3-1, Higashiyama-cho, Kobe, Hyogo 652-0042, Japan, Tel: +81-78-511-3131; Fax: +81-78-511-3138; E-mail: takeuchi_nobuhiro@kawasaki-hospital-kobe.or.jp

Received January 23, 2014; **Accepted** March 20, 2014; **Published** March 22, 2014

Citation: Takeuchi N, Nomura Y, Nishida Y, Maeda T, Tada H, et al. (2014) Endoscopic Submucosal Dissection for Early Gastric Cancer Complicated by Gastric Gland Heterotopia: A Case Report. J Clin Case Rep 4: 352. doi:10.4172/2165-7920.1000352

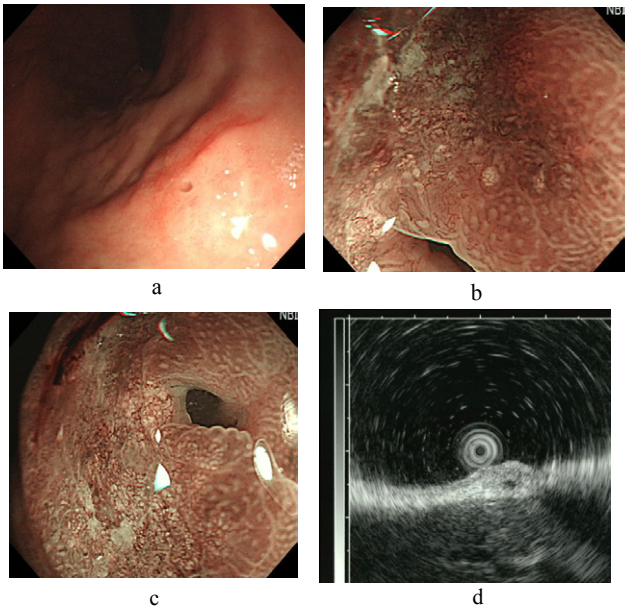

Figure 1: a. Gastroendoscopy revealed a small orifice near the anal side of the depressed lesion, which was considered to be gastric gland heterotopias. **b and c.** Narrow-band imaging endoscopy revealing irregular vascular patterns in the surface structures of the type 0-IIc lesion; this is consistent with the findings of gastric cancer. **d.** Endoscopic ultrasonography revealed that an anechoic lesion in the third layer was adjacent to a depressed lesion, suggesting that the depressed lesion had invaded the submucosal layer.

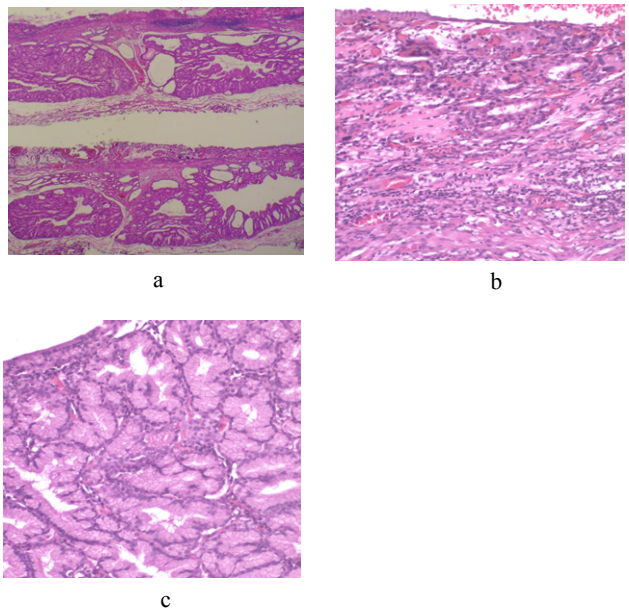

Figure 2: a. Pathological findings of the resected specimen reveal moderately differentiated tubular adenocarcinoma within the mucosal layer and multiple cystic dilated lesions in the submucosal layer (low power field). **b.** Moderately differentiated tubular adenocarcinoma within the mucosal layer is shown (high power field). **c.** The cystic dilated lesion in the submucosal layer are shown (high power field) (c).

or submucosal layers [4]. Its origin is classified as congenital [1] and acquired [4]; congenital GGH occurs because of the involvement of glands in the submucosal layer; however, acquired GGH occurs because of repeated inflammation and epithelial components involved in the submucosal layer. Currently, most researchers support the theory that GGH occurs because of acquired causes, such as repeated erosion and regeneration of the gastric mucosa, destruction of the mucosal lamina, and involvement of the regenerated gland in the mucosal layers through gaps in the mucosal lamina. Moreover, cancers may also arise because of prolonged inflammation.

In general, GGH takes diverse forms; therefore, it is usually difficult to diagnose GGH by radiography or gastroendoscopy. However, EUS is an efficient tool for diagnosing GGH; EUS reveals multiple cystic anechoic lesions in the third layer. The diagnostic accuracy of the invasion depth of early gastric cancer without ulcer lesions is reported to be 80%-100% in mucosal cancers and 74%-85% in submucosal cancers [5]. Multiple GGH is associated with the genesis of gastric cancer. However, there are some case reports on solitary GGH complicated with gastric cancer [6,7]. There is no established treatment of GGH, but lesions of >3 cm or cases that are difficult to confirm are surgically treated.

Assessment of the diagnostic depth of gastric cancer invasion complicated by GGH by EUS is sometimes difficult because GGH in the submucosal layer leads to misdiagnosis of the depth of gastric cancer invasion. Even in cases in which early gastric cancer could be diagnosed as mucosal cancer by endoscopy, these cases are diagnosed as submucosal cancer by EUS; therefore, there may exists a case in which preoperative diagnosis was submucosal cancer, and subsequently, when surgical resection was performed, postoperative pathological findings confirmed the case to be mucosal cancer [8]. Early gastric cancer complicated with GGH should be at first treated with ESD to avoid unnecessary surgical treatment, even when a massive submucosal invasion was suggested by EUS, although in our case the mucosal invasion was diagnosed by endoscopy.

Conclusion

Here we present a case in which early gastric cancer was complicated by GGH and was treated with ESD, although it was uncertain whether GGH in this case was associated with carcinogenesis. In this case, the presence of GGH made diagnosing the disease difficult because of the depth of invasion by EUS. Early gastric cancer accompanying GGH complicated the diagnosis of depth invasion; therefore, we ESD should be used to treat such case instead of other unnecessary surgical treatments.

References

1. Scott hwJr, Payne TP (1947) Diffuse congenital cystic hyperplasia of stomach clinically simulating carcinoma; report of a case. Bull Johns Hopkins Hosp 81: 448-455.
2. Oberman HA, Lodmell JG, Sower ND (1963) Diffuse heterotopic cystic malformation OF THE Stomach. N Engl J Med 269: 909-911.
3. Tchertkoff V, Wagner BM (1966) Diffuse cystic malformation of stomach. N Y State J Med 66: 2049-2052.
4. Iwanaga G, Furukawa H, Ishiguro S (1986) Histological observation of 102 cases with submucosal diffuse heteropathia of the stomach. SaishinIgaku 41: 2418-2426. [Article in Japanese]
5. Chonan A, Mishima T, Miyake N (2009) Endoscopic Ultrasonographic Diagnosis of Depth of Invasion of Early Gastric Cancer. Stomach and Intestine 44: 623-635.
6. Inoue S, Chonan A, Yuki T (1995) Gastric Glands Showing Characteristic Endoscopic Ultrasonographic Findings. A Case Report. Gastroenterological endoscopy 37: 2216- 2221.
7. Hara T, Hasui H, Moriguchi M, Kaji Y, Yosikawa Y (1994) Early Gastric Cancer Associated with Solitary Submucosal Heterotopic Gastric Glands, Report of a Case. Stomach and Intestine 29: 1089-1093.
8. Tamami Nakamura, TakaakiTsushimi, Toshiki Tanaka, Yoshihiro Takemoto, Eijiro Harada (2011) Multiple Early Gastric Cancers with Diffuse Cystic Malformation Mimicking Submucosal Invasion on Endoscopic Ultrasonography: Report of a Case. Yamaguchi Igaku 60:17.