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CASE REPORT

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Simultaneous laparoscopic hiatal hernia repair and pyloroplasty for a type 3 hiatal hernia with post-ESD pyloric stenosis for early gastric cancer

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Abstract

We present a case of early gastric cancer in the pylorus with a type 3 hiatal hernia, which was treated by endoscopic submucosal dissection (ESD). A 70-year-old man was referred to our hospital with a hiatal hernia. Endoscopy revealed early gastric cancer, and we performed an ESD adaptation at the pylorus. The ESD was successful, but post-ESD pyloric stenosis occurred. Symptoms of hiatal hernia worsened because of the pyloric stenosis. Laparoscopic hiatal hernia repair with Toupet fundoplication and Heineke-Mikulicz pyloroplasty was simultaneously performed. The postoperative course was good, and follow-up after discharge was uneventful. To our knowledge, there have been no reports in which laparoscopic hiatal hernia repair, fundoplication, and pyloroplasty were simultaneously performed for a substantial hiatal hernia with post-ESD pyloric stenosis.

KEYWORDS

fundoplication, gastric cancer, hiatal hernia

1 | INTRODUCTION

Hiatal hernia is a common disorder of diaphragmatic hernia.[1] Gastric cancer with esophageal hiatal hernia has been reported;[2-4] however, reports of early gastric cancer (EGC) treated with endoscopic submucosal dissection (ESD) in the pylorus, including a hiatal hernia are lacking. Because of the low invasiveness and almost no lymph node metastasis in EGC with an ESD adaptation, endoscopic treatment is recommended. However, additional gastrectomy may be required, depending on the pathological diagnosis.[5,6] Endoscopic resection with ESD adaptation has been widely used for treating EGC. ESD complications include bleeding, perforation, or stenosis. Stenosis is more likely to occur after ESD at a pyloric lesion.[7,8] Balloon dilation is used to treat stenosis, but has a risk of perforation.[9] We encountered a case of type 3 esophageal hiatal hernia and ESD-adaptive EGC at the pylorus. ESD resulted in pyloric stenosis, leading to exacerbation of reflux symptoms. The laparoscopic approach was eventually employed to simultaneously treat pyloric stenosis and type 3 hiatal hernia, with good short-term results. This approach is relatively new and may be useful for surgeons encountering similar cases with increasing endoscopic treatment of EGC.

2 | CASE PRESENTATION

A 70-year-old man presented with regurgitation and abnormal chest radiography. His medical history included

spastic coronary angina, prostate cancer (post-prostatectomy), and diabetes mellitus. Routine blood examination showed no abnormalities. Chest radiography revealed a gastric bubble on the diaphragm. Computed tomography showed that half of the stomach was prolapsed into the mediastinum (Figure 1). A type 3 esophageal hiatal hernia was, thus, diagnosed. Upper gastrointestinal endoscopy performed to exclude malignant lesions revealed two superficial depressed lesions. One lesion was a type 0-IIa-shaped, 10-mm tumor located at the lesser curvature and anterior wall of the antrum of the stomach. The other lesion was a type of 0-IIc 20-mm tumor located at the pylorus of the stomach. The pathological diagnosis by biopsy was adenocarcinoma (tub1 > tub2) for both lesions. Both were diagnosed as intramucosal lesions and indicated for ESD, but pyloric scar stenosis after ESD was a concern. When pyloric stenosis occurred, we planned to add pyloroplasty simultaneously with hernia repair, and ESD was performed subsequently. The resected tissue at the pylorus measured 40 × 26 mm, and the ulcer after excision occupied 7/8 laps at the pylorus (Figure 2). The antral resected tissue measured 35 × 20 mm. After ESD, pyloric stenosis occurred, causing abdominal pain in addition to reflux symptoms. After ESD, the endoscope was unable to pass the pylorus because of the stenosis (Figure 3). The 24-hours pH impedance monitor revealed 123 backflows in 1 day. The patient could eat soft meal, and laparoscopic hiatal hernia repair and pyloroplasty were performed 4 months after ESD.

Surgery was performed laparoscopically. Laparoscopic esophageal hiatal hernia repair (Toupet fundoplication), and laparoscopic pyloroplasty (Heineke-Mikulicz) were performed. Under general anesthesia, the patient was placed in a reverse Trendelenburg position with his legs apart. The ports were arranged as shown in Figure 4. First, esophageal hiatal hernia repair was performed. The stomach was reduced back into the peritoneal cavity gently, and the hernial orifice was sutured with 2-0 ethibond (Ethicon), and Toupet fundoplication was also performed. Subsequently, laparoscopic Heineke-Mikulicz pyloroplasty was performed. Another port was added, and intraoperative endoscopy was performed to evaluate fundoplication and pyloroplasty. The operation time was 315 minutes, with 20 mL blood loss. The operation and postoperative courses were uneventful. Oral intake of liquid food was started on postoperative day 2. Upper gastrointestinal series was performed on postoperative day 3, revealing no reflux of the cardia and good passage of the pylorus. The patient was discharged on postoperative day 8. The postoperative course was good,

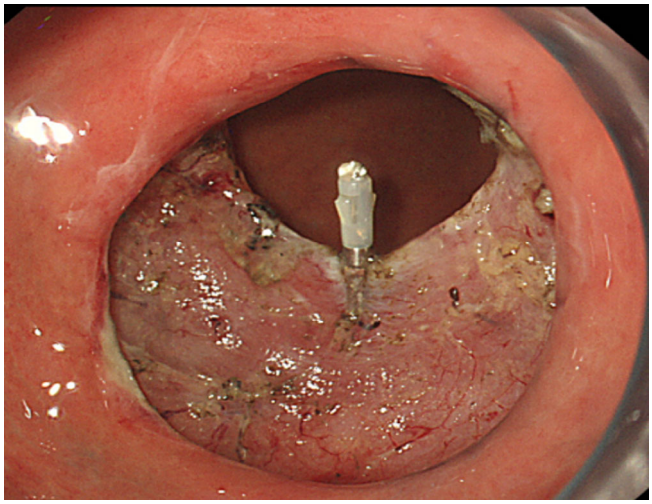

FIGURE 2 Endoscopy shows that post-endoscopic submucosal dissection ulcers occupied 7/8 laps at the pylorus

FIGURE 1 Computed tomography slices show that half of the stomach is prolapsed into the mediastinum

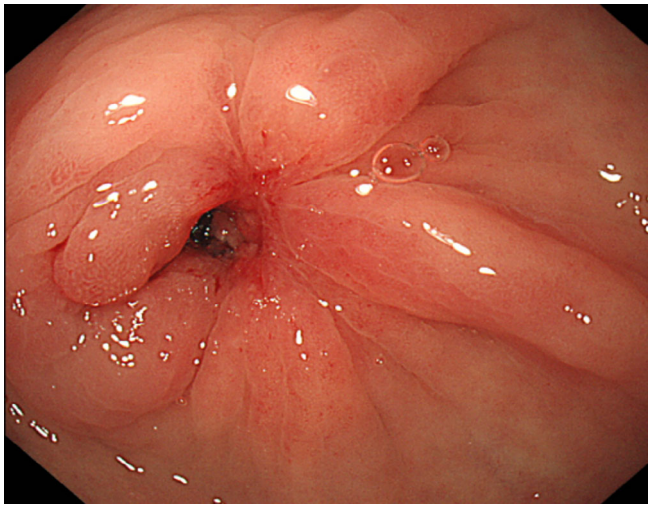

FIGURE 3 One month after endoscopic submucosal dissection, endoscopy reveals pyloric stenosis

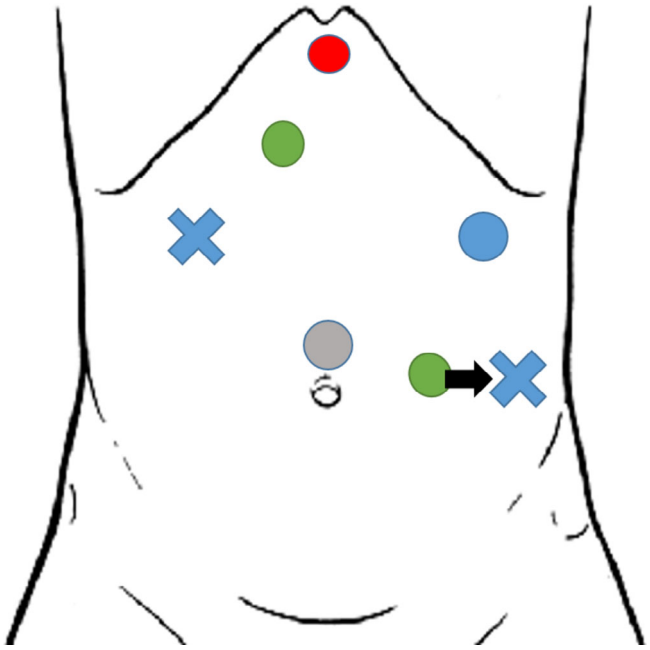

FIGURE 4 This figure shows port positions. The blue circle and cross are 12 mm ports. The green circles are 5 mm ports. The gray circle is a camera port. The red circle is a Nathanson liver retractor. Laparoscopic hiatal hernia repair and fundoplication were performed using four ports (circles). Then, an additional port was added, and one 5 mm port was changed to 12 mm port (cross); laparoscopic pyloroplasty was performed using five ports

with no symptoms associated with reflux or passage obstruction. One year after the operation, endoscopy showed no reflux esophagitis, no obstruction of passage through the pylorus, and no gastritis associated with bile reflux.

3 | DISCUSSION

Gastric cancer accompanied by esophageal hiatal hernia is relatively common and has been well described. Mimatsu et al. reported the clinicopathological characteristics, treatment, and outcomes of 14 patients with gastric cancer from the upside-down stomach through a paraesophageal hernia.[3] Hagiwara et al. reported on five patients who underwent laparoscopic gastrectomy for gastric cancer accompanied by huge hiatal hernia.[2] In this case, preoperative endoscopic examination revealed an EGC for an ESD adaptation. Endoscopic treatment or surgery must be selected according to the gastric cancer stage; hence, it is necessary to check for malignant diseases such as esophageal and gastric cancer before surgery for an esophageal hiatal hernia.

In this case, ESD was performed before the surgery, but post-ESD stenosis at the pylorus occurred. Stenosis reportedly occurs as a complication after ESD, with an incidence rate of 0.7%-1.9%. Scar stenosis due to ESD is known to occur more easily in the pylorus than in other sites.[6] The risk factors for post-ESD stenosis include the circumferential extent of mucosal defect of >75% of the circumference and a longitudinal extent of >5 cm.[7,10] Although stenosis may improve over time, conservative treatment for 6 months is often selected. Balloon dilation is performed for persistent and severe stenosis.[8,9] However, post-ESD stenosis of the pylorus needs a long time and many times for balloon dilation in comparison to stenosis of other organs; further, the risk of perforation is higher when the ESD scar extends to the duodenum.[9] Recently, per oral endoscopic pyloromyotomy (POP), also known as gastric per oral endoscopic myotomy (GPOEM), has been proposed as an alternative to surgical pyloroplasty for management of gastroparesis.[11] In this case, surgical pyloroplasty was selected because of scar stenosis after ESD. Further, steroid therapy, known to be effective in preventing stenosis after esophageal ESD,[12] is also effective after gastric ESD.[13] In this case, we tactically performed ESD first because pyloroplasty can be performed at the time of hernia repair if stenosis occurs, and gastrectomy, if EGC is not cured by ESD. However, steroids were not used during ESD, and their use may have reduced stenosis. The surgical invasion by additional pyloroplasty extended the operation time and required one more trocar, but showed good short-term results; hence, this treatment strategy was considered feasible. This approach should be taken into consideration when surgery is indicated in similar cases.

4 | CONCLUSIONS

We successfully treated a case of type 3 esophageal hiatal hernia with pyloric stenosis after ESD for EGC by simultaneously performing pyloroplasty and hiatal hernia repair. It is a practical and feasible approach for patients with pyloric stenosis and hiatal hernia.

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AUTHORSHIP DECLARATION

All persons who meet authorship criteria are listed as authors. All authors certify that they have participated sufficiently to take public responsibility for the content, including participation in the concept, design, analysis, writing, or revision of the manuscript. All authors are in agreement with the content of the manuscript. Furthermore, each author certifies that this material or similar

material has not been and will not be submitted to or published in any other publication before its appearance in the *Asian Journal of Endoscopic Surgery*.

AUTHORSHIP CONTRIBUTIONS

Category 1.

Conception and design of the study: Tetsushi Kubota, Idani Hitoshi; acquisition of data: Tetsushi Kubota, Idani Hitoshi; analysis and/or interpretation of data: Tetsushi Kubota, Hitoshi Idani.

Category 2.

Drafting the manuscript: Tetsushi Kubota, Idani Hitoshi; revising the manuscript critically for important intellectual content: Michihiro Ishida, Yasuhiro Choda, Kanyu Nakano, Yasuhiro Shirakawa, Shigehiro Shiozaki.

Category 3.

Approval of the version of the manuscript to be published (the names of all authors must be listed): Tetsushi Kubota, Hitoshi Idani, Michihiro Ishida, Yasuhiro Choda, Kanyu Nakano, Yasuhiro Shirakawa, Shigehiro Shiozaki.

CONSENT STATEMENT

Informed consent has been obtained from the patient.

DATA AVAILABILITY STATEMENT

All data generated or analysed during this case report are included in this published article.

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