

Endoscopic treatment of early esophageal cancer with decompensated cirrhosis and successful prevention of postoperative stenosis: A case report

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Abstract. The management of gastrointestinal tumors with decompensated cirrhosis is extremely challenging. Patients often present with poor basic condition and coagulation function, and nutritional deficiency. Furthermore, postoperative recovery is difficult and so the majority of patients refuse surgery. The present study reports the case of a 73-year-old man with decompensated cirrhosis and early esophageal cancer. At the discretion of the patient and their family, a simultaneous approach was used to treat esophagogastric varices and perform a mucosal dissection of the early esophageal cancer via endoscopy. Post-surgery, multiple polyglycolic acid sheets were attached to the esophageal dissection wound. At >2 months post-surgery, an endoscopic re-examination of the patient showed that the esophageal mucosa had healed well, and there was no resistance detected via ordinary endoscopy. The main objective of the present study was to highlight the feasibility and safety of endoscopic treatment for patients with decompensated liver cirrhosis complicated with early esophageal cancer, and to provide a new treatment strategy for patients at high risk of esophageal stenosis after endoscopic mucosal dissection.

Introduction

Endoscopic mucosal dissection has become the first-choice treatment for early esophageal cancer. Any invasive exercises are challenging in patients with decompensated cirrhosis due to poor coagulation and a poor condition. Liver cirrhosis has been shown to be a risk factor for esophageal cancer surgery;

however, endoscopic treatment appears to be safe, particularly in patients without severe liver dysfunction (1). Simultaneously, stenosis of early esophageal cancer after endoscopic mucosal dissection is a noteworthy problem. A circumferential mucosal defect more than three-fourths the circumference of the esophagus, mucosal defects longer than 40 mm and an infiltration depth greater than the lamina propria of the mucosa are independent risk factors for stenosis after endoscopic mucosal dissection (2-4). The patient in the present case report had independent risk factors for the postoperative stenosis of esophageal cancer, including an esophageal dissection circumference $\geq 3/4$, cirrhosis with esophageal and gastric varices, ascites and other complications, which increased the difficulty of this treatment. The main objective of this study was to explore the feasibility of endoscopic treatment of decompensated cirrhosis with early cancer and to provide a protocol for the prevention of stenosis after endoscopic mucosal dissection.

Case report

A 73-year-old man was admitted to Xiangyang Central Hospital (Xiangyang, China) in April 2022 due to swallowing difficulties that had persisted for the past 1 month. At the end of the first month, progressive dysphagia gradually developed without obvious induction, particularly while consuming dry rice and rough food. No signs of obvious obstruction were observed while consuming liquid or semi-liquid food. He reported no symptoms of pharyngeal pain, a burning sensation behind the sternum, vomiting after eating, fatigue, abdominal pain, abdominal distension, diarrhea, nausea, acid regurgitation or belching. Gastroscopy at Xiangyang Central Hospital demonstrated an irregularly raised mass 30 cm away from the incisors, suspected to be esophageal cancer. The patient was then admitted to the Department of Surgery of Tongji Hospital (Wuhan, China), where an endoscopic examination was conducted (Fig. 1A). The examination showed a rough and uneven esophageal mucosa, with varicose veins at the fundus eminence. The patient suffered from chronic erosive gastritis [grade II, Kyoto classification (5)] and chronic atrophic gastritis (grade I). Biopsy specimens from the esophagus and the gastric angle confirmed high-grade squamous intraepithelial neoplasia with focal suspicious infiltration and (horn) gastric

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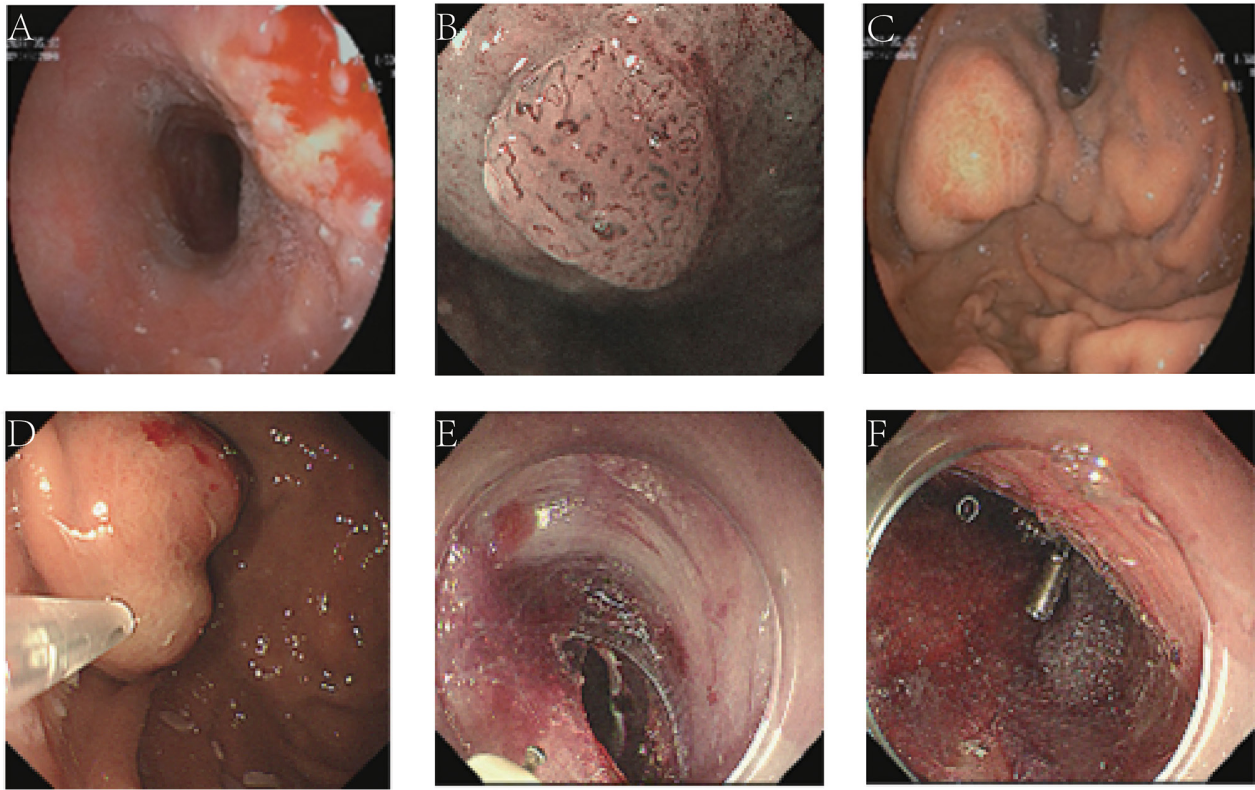


Figure 1. Preoperative examination and surgical procedure. (A) Image taken from 28-33 cm along in the patient's esophagus. The mucous membrane was rough and uneven, with a little yellow moss attached, and was brittle and bled easily. (B) Gastric varices. (C) Under magnification endoscope, the intra-epithelial papillary capillary loop mainly showed B1 type, local B2 type and suspicious B3 type [magnifying endoscopic classification (24)] vessels in the uplift. (D) Sandwich method for gastric varices tissue glue injection. (E) Mucosal dissection of early esophageal cancer, complete dissection and successful operation. (F) Post-surgery, multiple pieces of polyglycolic acid sheet were attached to the wound and fixed with titanium clips.

mucosa showing chronic inflammatory changes with mild intestinal metaplasia. Thereafter, the patient was transferred to the Department of Gastroenterology for further treatment.

The patient's medical history reported hepatitis B viral cirrhosis. A physical examination revealed a body temperature of 36.3°C (normal range, 36.2-37.2°C), a heart rate of 72 beats/min (normal range, 60-100 beats/min), a blood pressure of 159/89 mmHg (normal ranges, systolic blood pressure ≥ 90 and < 140 mmHg; diastolic blood pressure ≥ 60 and < 90 mmHg) and a respiratory rate of 20 breaths/min (normal range, 12-20 breaths/min). The abdomen was flat and soft with audible bowel sounds 2-3 times/min. The liver, spleen and the mass were not palpable, and there was no tenderness present. There was no edema or restriction in the movement of either of the lower limbs. Serological examination revealed hypersplenism, mild liver dysfunction, mild coagulation dysfunction, active hepatitis B virus replication and no abnormalities in renal function or electrolyte levels (Table I). Additionally, abdominal Doppler ultrasound showed signs of liver cirrhosis and intrahepatic bile duct stones. Furthermore, contrast-enhanced chest computed tomography (CT) (Fig. 2) revealed micronodules in the left lower lobe of the lung, bilateral emphysema, and mediastinal lymph nodes increased and some of them enlarged. Based on a consultation with imaging experts, it was considered that the left lower lung lobe likely had a benign lesion, and dynamic observations were planned during the follow-up. CT-hepatic portal vein angiography demonstrated cirrhosis, splenomegaly, lower esophageal varices



Figure 2. Preoperative CT. On day 5 after hospitalization, contrast-enhanced chest CT indicated micronodules in the left lower lobe of the lung and bilateral emphysema. CT, computed tomography.

and minor ascites. Magnification endoscopy (Fig. 1B and C) revealed esophageal grade 0-IIb+IIa lesions [Paris endoscopic classification (6)] indicating early cancer (suspected deep infiltration of submucosa), gastric varices (severe) and chronic atrophic gastritis (grade I). After repeated communication with the patient and their family, based on the results of the

Table I. Laboratory findings of the first visit in May 2022.

Laboratory test	Actual value (normal range)
White blood cell count, $10^9/l$	2.87 (3.50-9.50)
Red blood cell count, $10^{12}/l$	2.54 (4.30-5.80)
Platelet count, $10^9/l$	55.0 (125.0-350.0)
Hemoglobin, g/l	78.0 (130.0-175.0)
Prothrombin time, sec	20.7 (11.5-14.5)
Internationalization standard value	1.75 (0.80-1.20)
Activated partial thromboplastin time, sec	55.3 (29.0-42.0)
α -fetal protein, ng/ml	12.79 (≤ 7.00)
Aspartate aminotransferase, U/l	60 (≤ 40)
Alanine aminotransferase, U/l	42 (≤ 41)
HBV-DNA, IU/ml	2.22×10^6
HBV, hepatitis B virus.	

Table II. Laboratory findings of the subsequent visit in September 2022.

Laboratory test	Actual value (normal range)
White blood cell count, $10^9/l$	4.77 (3.5-9.5)
Red blood cell count, $10^{12}/l$	3.95 (4.30-5.80)
Platelet count, $10^9/l$	131.0 (125.0-350.0)
Hemoglobin, g/l	104.0 (130.0-175.0)
Aspartate aminotransferase, U/l	52 (≤ 40)
Alanine aminotransferase, U/l	40 (≤ 41)
HBV-DNA, IU/ml	$< 1.00 \times 10^2$
HBV, hepatitis B virus.	

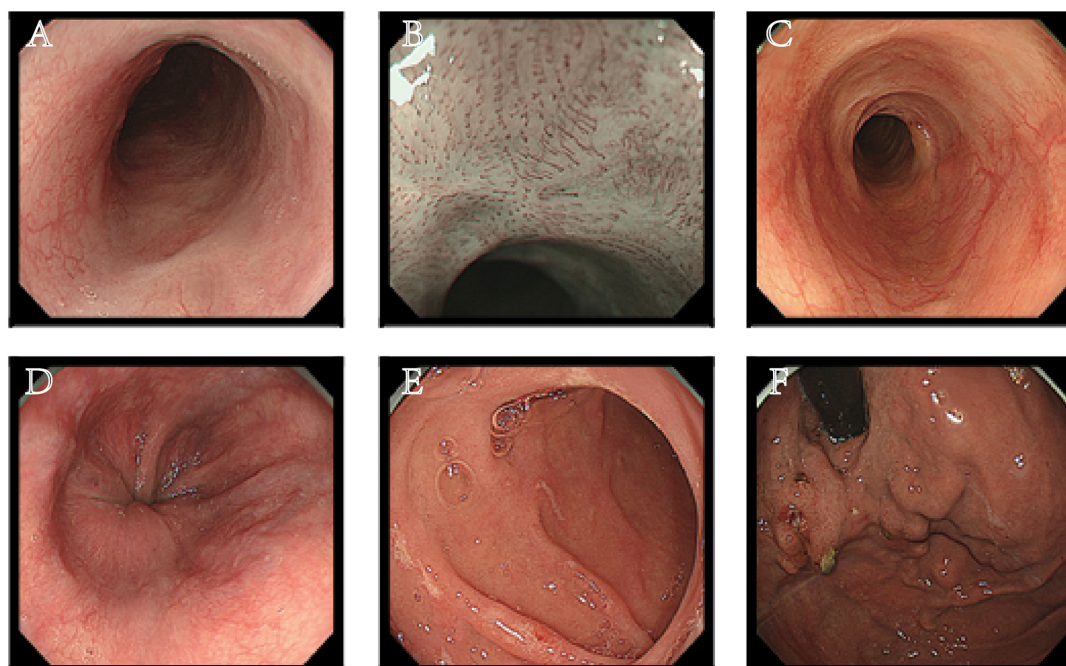


Figure 3. Endoscopic reexamination in September 2022. (A) 25 cm from the start of the esophagus. (B) Magnifying endoscope: 30 cm from the start of the esophagus. (C) 30 cm from the start of the esophagus. (D) 40 cm from the start of the esophagus. (E) Stomach body. (F) Under the cardia. The esophageal mucosa had healed well, and there was no resistance on ordinary endoscopy. The esophagus and stomach showed postoperative scar changes.

magnification endoscopy, the patient was prescribed open surgical treatment. However, this was declined owing to the patient's age, and therefore, endoscopic surgery was planned. Since esophageal and gastric varices were present, the patient was at a high risk of stenosis after endoscopic mucosal dissection for early esophageal cancer, and so with consent, both treatment for endoscopic varices and polyglycolic acid (PGA) sheet attachment for the prevention of esophageal stenosis were scheduled. Endoscopic treatment was performed at the end of May 2022 (Fig. 1D-F). Tissue glue injection for the esophageal and gastric varices, and mucosal dissection for the early esophageal cancer, were successively performed. Simultaneously, multiple PGA sheets were attached to the wound on the esophageal dissection surface and fixed with titanium clips. The clips did not need to be removed after surgery, and would fall off automatically after a period of time. The patient was treated with proton-pump inhibitors (for 4 weeks), somatostatin analogs (for 7 days), hemostasis and fluid rehydration (for 3 days), and was also treated for a fever on the first day after the operation. Anti-infection therapy (for 4 days) was also initiated. On the third day post-surgery, the patient's body temperature gradually decreased and a liquid diet was introduced. On the sixth day post-surgery, the pathological results revealed that the esophageal squamous cell carcinoma (high to moderate differentiation) had invaded the submucosa of the esophageal wall (submucosal infiltration depth of ~1.1 mm); no tumor thrombus was found in the gastric mucosa and submucosal vessels, with negative horizontal and vertical margins. Treatment with surgery or chemoradiotherapy was recommended; however, the patient and their family refused this treatment. The patient was discharged on the seventh day. Oral hormones (which were discontinued within 12 weeks of discharge) and hepatitis B virus replication inhibitors (to be taken for life) with supplementation for stomach lining protection (for 12 weeks) with calcium supplementation (for 12 weeks), and anticoagulant therapy (for 12 weeks), were prescribed.

Gastroscopy was performed in September 2022 (Fig. 3). There was no resistance on ordinary endoscopy, the esophageal mucosa had healed well and no residual polyglycolic acid sheet was found. There were no gastrointestinal bleeding, fracture, infection, new-onset diabetes or other complications during medication administration. Reexamination of the serology showed that the white blood cell count, red blood cell count and platelet count in the blood routine test results were close to the normal value ranges, the liver function was improved and hepatitis B virus replication was low (Table II). The patient will continue to undergo follow-up.

Discussion

Cirrhosis is considered an independent risk factor for esophageal cancer surgery. Particularly for patients with decompensated cirrhosis who have poor coagulative function and poor esophageal vascular conditions, surgical treatment is challenging. Studies have shown that patients with liver cirrhosis are more likely to have pulmonary complications, ascites and anastomotic leakage or fistula within 30 days of surgery for esophageal cancer (7,8). Furthermore, some studies report that patients with cirrhosis have a higher mortality rate after surgery than those without cirrhosis (9,10). However, use of endoscopic treatment

for liver cirrhosis with early esophageal cancer appears to be evidentially safer (11). To the best of our knowledge, no studies have reported a patient experiencing endoscopic mucosal dissection-related esophageal perforation, postoperative bleeding or death (1,12). In patients with gastroesophageal varices, the varices must be treated before endoscopic mucosal dissection, as serious adverse events have been reported to occur after endoscopic mucosal dissection (13). Endoscopic treatment of liver cirrhosis complicated by early esophageal or gastric cancer has been reported to be safe (14,15). Another retrospective study showed no significant difference in the incidence of complications after endoscopic mucosal dissection and no deterioration of liver function after endoscopic mucosal dissection in liver cirrhosis complicated with gastric tumors in different liver function groups (16). However, further research with more cases is needed to corroborate this.

Currently, there is no standard prevention program regarding stenosis after endoscopic mucosal dissection for early esophageal cancer. In China, most doctors prefer to use oral hormones to prevent stenoses. However, previous studies (17,18) have shown that oral steroids have a minute effect, which is not sufficient for patients with high-risk factors for stenosis, especially for patients with a defect $\geq 3/4$ the circumferential range of the esophagus after esophagectomy. A case study of a 73-year-old man from South Korea in which PGA was used to prevent strictures after esophageal endoscopic submucosal dissection surgery reported no evidence of recurrence at the 1-year follow-up (19). In addition, a Japanese study (20) showed that the patients with circumferential range more than 1/2 had a postoperative stenosis rate of only 1/13. Another study showed that PGA combined with fibrin glue had a stenosis rate of 3/8 in patients with a circumferential range $>3/4$ (21). Moreover, a 2017 retrospective study (22) showed that the stenosis rate of PGA combined with fibrin glue was 3/33. A domestic prospective study (23) in 2018 showed that the stenosis rate of PGA combined with esophageal stents was 7/34, which was significantly lower than the 15/32 recorded for the stent group. Consequently, these studies have shown that PGA has a good effect on the prevention of esophageal cancer strictures after endoscopic submucosal dissection. Furthermore, PGA can promote wound healing and additionally serve as a physical support for the postoperative esophagus. Due to the physical properties of PGA, it degrades in ~1 month, eliminating the need for a second endoscopic resection, which significantly reduces endoscopic pain and surgical costs for the patients.

In conclusion, the present study reports the successful treatment a patient with decompensated cirrhosis and early esophageal cancer, and the prevention of stenosis after endoscopic mucosal dissection, resulting in a considerable improvement in the patient's quality of life. Close follow-up of the patient will continue.

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Availability of data and materials

All data generated or analyzed during this study are included in this published article.

Authors' contributions

WT collected patient data and designed the study. ML and XXF performed the surgery. XXF participated in the analysis and interpretation of the data. WT and XXF both contributed to manuscript drafting, writing and final correction of the manuscript. WT, ML and XXF read and approved the final manuscript, and confirm the authenticity of all the raw data.

Ethics approval and consent to participate

The study was conducted according to the guidelines of the Declaration of Helsinki and was approved by the Ethics Committee of Huazhong University of Science and Technology (Wuhan, China).

Patient consent for publication

The patient provided written informed consent for the publication of this case report and all accompanying images.

Competing interests

The authors declare that they have no competing interests.

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