Effectiveness of an orally administered steroid gel at preventing restenosis after endoscopic balloon dilation of benign esophageal stricture

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Abstract
Background: Restenosis occurs in 10% to 30% of patients after endoscopic balloon dilation (EBD) of benign esophageal stricture. This study aimed to investigate whether an orally administered steroid/gel mixture would inhibit restenosis after EBD for benign esophageal stricture.

Methods: This retrospective analysis included patients with benign esophageal stricture treated with EBD at the Peking University Third Hospital, China (September 2005 to October 2017). The patients were divided into an EBD alone group (EBD only) and EBD/OHA group (EBD plus oral hydrocortisone sodium succinate/aluminum phosphate gel mixture). Patients were followed-up for 3 months. EBD was repeated for persistent dysphagia. The outcome measures included the restenosis rate and the number of additional EBD sessions required during follow-up. Any side effects were noted.

Results: Thirty-two patients were included (EBD alone group, n=12; EBD/OHA group, n=20). Baseline demographic and clinical characteristics (including age, sex, etiology, stricture location, and diameter of narrowest segment) did not differ between groups. Restenosis rate was significantly lower in the EBD/OHA group than in the EBD alone group (5/20, 25.0% vs 8/12, 66.7%; P=.025). The median number of EBD sessions required was numerically lower in the EBD/OHA group (median, 1.0; interquartile range, 1.0–1.8) than in the EBD alone group (median 2.0, interquartile ranged 1.0–2.5), but statistical significance was not attained (P=.071). The only side effects occurred in the EBD/OHA group: hypokalemia in 4/20 (20.0%) and apathy/somnolence in 1/20 (5.0%).

Conclusions: An orally administered steroid/gel mixture might reduce the incidence of restenosis after EBD for benign esophageal stricture, but it requires confirmation by prospective trials.

Abbreviations: EBD = endoscopic balloon dilation, EGD = esophagogastroduodenoscopy, ESD = endoscopic submucosal dissection, GERD = gastroesophageal reflux disease, IQR = interquartile range.

Keywords: benign esophageal stricture, endoscopic balloon dilation, restenosis, steroid gel

1. Introduction

Although benign esophageal stenosis is an uncommon condition with a reported incidence of 1.1 per 10,000 person-years,1 those affected can experience unpleasant symptoms such as dysphagia, heartburn, choking, and coughing. Most cases of esophageal stricture are secondary to gastroesophageal reflux disease (GERD), but other causes include surgery, circumferential endoscopic dissection for early esophageal cancer, radiotherapy, corrosive substances, Schatzki ring, and certain drugs.2–4 The incidence of benign esophageal stenosis increases with age, and risk factors associated with esophageal stricture include prior dysphagia, GERD, hiatus hernia, peptic ulcer disease, and high level of alcohol intake.1

A variety of techniques have been used for the treatment of benign esophageal stenosis, including mechanical dilation of the esophagus, stent placement, incisional therapy, and the local injection of anti-inflammatory or anti-fibrotic agents.1,5,6 However, the management of benign esophageal strictures usually consists of lifelong antireflux measures and periodic mechanical esophageal dilation.5,6 Endoscopic balloon dilation (EBD) is usually the first choice technique for esophageal dilation, and most patients obtain relief of their symptoms after 1 to 5 treatments.5,7–10 However, 10% to 30% of patients are refractory to EBD and require repeated dilatations.3,7,11 Since repeated dilations are both risky for the patient and costly, it will
be important to identify novel and effective methods for preventing esophageal restenosis after EBD.

Steroids can potentially inhibit stricture formation by reducing collagen synthesis and promoting collagen breakdown. Several studies have demonstrated that prophylactic local injection of steroid is effective at reducing the occurrence of post-EBD restenosis.\textsuperscript{12-15} However, steroid injections are associated with a theoretical risk of esophageal perforation and local infection.\textsuperscript{11,16} In addition, the injection technique is complex, increasing the technical difficulty of the EBD procedure. The development of a new method for applying a steroidal drug to a stricture that avoids the need for injection would potentially improve the management of benign esophageal stenosis.

We hypothesized that an innovative strategy utilizing an orally administered mixture of hydrocortisone sodium succinate and aluminum phosphate gel would be effective at preventing restenosis after EBD, as well as being associated with lower risks of perforation and easier operation than injection of steroids. Therefore, the aim of our study was to investigate the feasibility and effectiveness of using an orally administered mixture of hydrocortisone sodium succinate and aluminum phosphate gel to reduce the occurrence of restenosis after EBD for benign esophageal stricture.

2. Patients and methods

2.1. Study design and patients

This was a retrospective analysis of consecutive adult patients with a diagnosis of benign esophageal stricture treated with EBD at the Department of Gastroenterology and Hepatology, Peking University Third Hospital, China between September 2005 and October 2017.

The inclusion criteria were:
(1) age ≥18 years;
(2) symptoms of dysphagia;
(3) endoscopy confirmed the presence of esophageal stenosis through which the body of a standard per oral gastroscope was unable to pass;
(4) biopsy did not show any evidence of malignant tumor; and
(5) EBD was used as the treatment.

The exclusion criteria were:
(1) endoscopic therapy was contraindicated, for example, due to severe cardiopulmonary insufficiency or coagulation system dysfunction;
(2) EBD refused by the patient;
(3) pathology suggested the presence of a malignant tumor at the site of the stenosis;
(4) complications during or after operation (not including restenosis) that required further treatment;
(5) contraindication to glucocorticoid drugs;
(6) administration of other treatments, such as additional local surgery or radiotherapy, during the follow-up period;
(7) data required for the analysis were missing; or
(8) lost to follow-up.

The study was approved by the Institutional Review Board of Peking University Third Hospital (no. M2016171). All patients provided informed written consent for their treatments. Patient consent for inclusion in this analysis was waived due to the retrospective study design.

2.2. Allocation of patients to the study groups

During the early stage of our study period (up to October 2014), our hospital adopted EBD as the standard treatment method for benign esophageal stenosis and did not utilize any additional methods aimed at preventing restenosis. During the later stage of the study (October 2014 onward), patients treated with EBD also received orally administered steroid/gel mixture (see below) as a potential preventative measure against scarring and restenosis. This was based on the clinical observation that the steroid/gel mixture was effective at inhibiting stenosis after endoscopic submucosal dissection (ESD). Therefore, the patients in this study were divided chronologically into 2 groups: those who were treated with EBD alone (EBD alone group) and those who received both EBD and therapy with oral steroid/gel mixture (EBD/OHA group). Patients in both groups were treated by conventional EBD in accordance with standard clinical practice.\textsuperscript{17} Representative images obtained during endoscopic dilation in patients in the EBD/OHA and EBD alone groups are shown in Figures 1 and 2, respectively.

2.3. Oral administration of the steroid/gel mixture

For patients in the EBD/OHA group, oral administration of the steroid/gel mixture was initiated the day after operation and continued for 8 weeks. The steroid/gel mixture consisted of hydrocortisone sodium succinate for injection (50mg/vial; Tianjin Biochemical Pharmaceutical Co Ltd, Tianjin, China) and aluminum phosphate bagged gel preparation (20g/bag; Boryung Pharm Co Ltd, Seoul, South Korea). The steroid/gel mixture was administered 4 times/d, and the dose of hydrocor-
tisone sodium succinate was gradually tapered during the 8-week administration period (50mg qid for 2 weeks, 25mg qid for 2 weeks, and 12.5mg qid for 4 weeks) whereas the aluminum phosphate dosage was kept constant (20mg qid). The hydrocortisone sodium succinate and aluminum phosphate gel were evenly mixed before administration. Each patient was asked to take the medication on an empty stomach, remain in bed for 30 minutes after oral administration of the steroid/gel mixture, and abstain from food or drink for 2 hours after taking the medication.

2.4. Baseline demographic and clinical characteristics

The following information was retrieved from the medical records: patient age, patient sex, history of previous EBD, stricture etiology (eg, previous surgery, previous ESD, GERD, or caustic), dysphagia symptom severity (grade I: occasional dysphagia but able to swallow soft food; grade II: able to swallow semi-liquid food; grade III: able to swallow solid food; grade IV: unable to swallow solids or liquids), stricture location (upper, middle, or lower esophagus), stricture diameter at the narrowest segment, and stricture diameter after EBD.

2.5. Follow-up and outcome measures

The patients were followed-up for 3 months. Esophagogastro-duodenoscopy (EGD) was performed on demand during the follow-up period if a patient complained of dysphagia, and the presence and extent of any restenosis were evaluated (including measurement of the esophageal diameter). EBD was performed again if the patient was deemed to have restenosis. If a patient experienced no symptoms of dysphagia during the follow-up
period, EGD was performed 12 weeks after the original EBD to evaluate whether a stricture was present. A stricture (ie, restenosis) was defined as a difficulty in swallowing solids or an inability to pass an endoscope (9.2 mm in diameter) at EGD. The dysphagia symptom severity was also evaluated at follow-up. The outcome measures were the restenosis rate after EBD, dysphagia symptom severity, number of additional EBD sessions required during the 3-month follow-up period, diameter of the restenosis and extent of the restenosis (none, mild, moderate, or severe).

2.6. Safety analysis

Blood pressure, blood glucose, and serum electrolytes were measured at all follow-up visits, and any side effects were recorded.

2.7. Statistical analysis

The analysis was performed using SPSS 23.0 (IBM Corp, Armonk, NY). All data were assessed for normality using the Kolmogorov–Smirnov test. Normally distributed data are presented as the mean ± standard deviation (range) and were compared between groups using Student t test. Non-normally distributed data are presented as the median (interquartile range [IQR]) and were compared between groups using Fisher exact test. A P-value < .05 was considered to represent a statistically significant difference.

3. Results

3.1. Patient enrolment

Among 36 patients who underwent EBD in our hospital between September 2005 and October 2017, 4 were excluded (underwent additional surgery during follow-up because a local malignant tumor could not be excluded, n = 1; underwent conservative treatment with stent placement because the morphologic appearance during endoscopy could not exclude a malignant tumor, n = 1; underwent surgical therapy due to gastrointestinal perforation during follow-up, n = 1; and lost to follow-up, n = 1). Therefore, 32 patients were included in the final analysis.

3.2. Baseline demographic and clinical characteristics

The baseline characteristics of the patients in the 2 groups are presented in Table 1. The 32 patients (25 males and 7 females) included in the study had a mean age of 66.1 ± 10.3 years (range, 41–86 years). The etiology of the esophageal stricture was most
commonly previous surgery (17/32, 53.1%) or previous ESD (9/32, 28.1%), with GERD (5/32, 15.6%) and caustic stricture (1/32, 3.1%) responsible for only a minority of cases. The stenosis was most frequently located in the lower esophagus (14/32, 43.8%), followed by the middle esophagus (10/32, 31.3%) and upper esophagus (8/32, 25.0%). The dysphagia symptom severity was grade IV in 18 cases (56.3%), grade III in 8 cases (25.0%), and grade II in 6 cases (18.8%). Esophagography showed that the diameter of the narrowest region of stenosis ranged from 1.5 to 7.0 mm with a median (IQR) value of 3.0 (2.0, 4.0) mm. After dilation, the esophageal diameter ranged from 10.0 to 15.0 mm with a median (IQR) value of 13.5 (11.0, 15.0) mm.

The EBD alone group consisted of 12 patients (11 males and 1 female) aged 65.3 ± 12.4 years (range, 41–85 years), and the EBD/OHA group contained 20 patients (14 males and 6 females).

Figure 2. Representative images obtained during and after EBD of a benign esophageal stricture. This 59-year-old male was not treated with the steroid/gel mixture and so was allocated to the EBD alone group. (A) Esophagography of the esophagus showing stenosis in the lower esophagus. (B and C) Endoscopic views obtained during the process of balloon dilation of the esophagus. (D) Endoscopic view after balloon dilation showing an unobstructed esophagus. (E) Esophagography after EBD demonstrated restenosis of the esophagus at 44 days after the original EBD. (F) Endoscopic view showing restenosis of the esophagus at 44 days after the original EBD. EBD = endoscopic balloon dilation.
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**Table 1**
Baseline demographic and clinical characteristics of the study participants.

| Characteristic          | EBD/OHA group (n = 20) | EBD alone group (n = 12) | P   |
|-------------------------|------------------------|--------------------------|-----|
| Sex, n (%)              |                        |                          |     |
| Male                    | 14 (70.0%)             | 11 (91.7%)               | .212|
| Female                  | 6 (30.0%)              | 1 (8.3%)                 |     |
| Age, yr, mean ± SD      | 66.5 ± 9.1             | 65.3 ± 12.4              | .761|
| Age distribution, yr, n (%) |                    |                          |     |
| <50                     | 0                      | 1 (8.3%)                 | .776|
| 50–59                   | 5 (25.0%)              | 3 (25.0%)                |     |
| 60–69                   | 7 (35.0%)              | 3 (25.0%)                |     |
| 70–79                   | 6 (30.0%)              | 3 (25.0%)                |     |
| ≥80                     | 2 (10.0%)              | 2 (16.7%)                |     |
| Etiology, n (%)         |                        |                          |     |
| Surgery                 | 9 (45.0%)              | 8 (66.7%)                | .151|
| ESD                     | 8 (40.0%)              | 1 (8.3%)                 |     |
| GERD                    | 3 (15.0%)              | 2 (16.7%)                |     |
| Caustic                 | 0                      | 1 (8.3%)                 |     |
| Dysphagia symptom grade, n (%) |                  |                          |     |
| I                       | 0                      | 0                        | 1.000|
| II                      | 4 (20.0%)              | 2 (16.7%)                |     |
| III                     | 5 (25.0%)              | 3 (25.0%)                |     |
| IV                      | 11 (55.0%)             | 7 (58.3%)                |     |
| Stricture location, n (%) |                      |                          |     |
| Upper esophagus         | 5 (25.0%)              | 3 (25.0%)                | .896|
| Middle esophagus        | 7 (35.0%)              | 3 (25.0%)                |     |
| Lower esophagus         | 8 (40.0%)              | 6 (50.0%)                |     |
| Diameter of stricture, mm, median (IQR) | 3.0 (2.0, 3.9) | 3.0 (3.0, 4.8) | .144|
| Diameter after dilation, mm, median (IQR) | 13.5 (12.0, 15.0) | 12.0 (10.5, 15.0) | .146|

**Table 2**
Comparison of outcome measures between the study groups.

| Outcome measure          | EBD/OHA group (n = 20) | EBD alone group (n = 12) | P   |
|--------------------------|------------------------|--------------------------|-----|
| Restenosis, n (%)        | 5 (25.0%)              | 8 (66.7%)                | .025|
| Dysphagia symptom grade during follow-up, n (%) |                  |                          |     |
| I                       | 15 (75.0%)             | 3 (25.0%)                | .030|
| II                      | 2 (10.0%)              | 3 (25.0%)                |     |
| III                     | 1 (5.0%)               | 2 (16.7%)                |     |
| IV                      | 2 (10.0%)              | 4 (33.3%)                |     |
| Degree of restenosis, n (%) |                      |                          |     |
| None                    | 15 (75.0%)             | 4 (33.3%)                | .011|
| Mild                    | 0                      | 4 (33.3%)                |     |
| Moderate                | 3 (15.0%)              | 1 (8.3%)                 |     |
| Severe                  | 2 (10.0%)              | 3 (25.0%)                |     |
| Diameter of restenosis, mm, median (IQR) | 11.0 (5.3, 12.0) | 6.5 (2.8, 9.0) | .005|
| Number of additional EBD sessions, median (IQR) | 1 (1, 1.8) | 2 (1, 2.5) | .071|

**EBD** = endoscopic balloon dilation, **ESD** = endoscopic submucosal dissection, **GERD** = gastroesophageal reflux disease, **IQR** = interquartile range, **OHA** = oral administration of hydrocortisone sodium succinate and aluminum phosphate gel.

Aged 66.5 ± 9.1 years (range, 54–86 years) old. Previous surgery was the most common cause of esophageal stricture in both the EBD/OHA and EBD alone groups (45.0% and 66.7%, respectively), whereas GERD accounted for less than 20% of cases in both groups (Table 1). There were no significant differences between the 2 groups in patient age, patient gender, stricture etiology, dysphagia symptom severity, stricture location, diameter of the stricture at the narrowest region, or esophageal diameter after balloon dilation (Table 1).

Seven of the patients in the EBD/OHA group (35%) had undergone previous dilation therapy at our hospital, compared with none in the EBD alone group. Among these 7 patients in the EBD/OHA group, previous endoscopic dilation therapy was performed twice in 2 patients, 5 times in 2 patients, 7 times in 2 patients, and 8 times in 1 patient. Thus, patients in the EBD/OHA group had received a median (IQR) of 0 (0, 4.3) endoscopic dilatations previously, compared with none in the EBD alone group (P = .023).

### 3.3. Outcome measures

All 32 patients were followed-up for at least 3 months (the follow-up duration ranged from 3 to 59 months, although only data for the first 3 months were included in the present analysis). A total of 13 patients (40.6%) were confirmed to have restenosis on repeat gastroscopy during follow-up (at 0.5–2 months). As shown in Table 2, the postprocedural esophageal restenosis rate was significantly lower in the EBD/OHA group than in the EBD alone group (5/20 patients, 25.0% vs 8/12 patients, 66.7%; P = .025). Median stenosis diameter during re-examination ranged from 2 to 15 mm in the EBD/OHA group and 2 to 10 mm in the EBD alone group. Furthermore, the median diameter of the stricture during re-examination was significantly larger in the EBD/OHA group than in the EBD alone group (11.0 [5.3, 12.0] vs 6.5 [2.8, 9.0] mm; P = .005), and the proportion of patients with severe stenosis was significantly lower in the EBD/OHA group than in the EBD alone group (2/20 patients, 10.0% vs 3/12 patients, 25.0%; P = .011). Consistent with the above observations, grade IV dysphagia was reported in fewer patients in the EBD/OHA group than in the EBD alone group (2/20 patients, 10.0% vs 4/12 patients, 33.3%; P = .030).

All 5 patients in the EBD/OHA group who developed restenosis received additional EBD treatment (1 EBD session in 3 patients and 2 EBD sessions in 2 patients). Among the 8 patients in the EBD alone group who developed restenosis, 3 were lost to follow-up after 3 months and did not undergo repeat EBD; the remaining 5 patients received additional EBD therapy (1 EBD session in 4 patients and 2 EBD sessions in 1 patient). The median (IQR) number of repeat EBD procedures during the follow-up period was 1 (1, 1.8) in the EBD/OHA group, which was numerically lower than the value of 2 (1, 2.5) in the EBD alone group; however, statistical significance was not attained (P = .071; Table 2).

Since 7 of the 20 patients in the EBD/OHA group had undergone endoscopic dilation treatment before inclusion in the study, an additional analysis was performed to determine whether the incidence of restenosis during the 3-month follow-up differed between those in the EBD/OHA group with a previous history of endoscopic dilation and those without. The restenosis rate did not differ significantly between patients who had received endoscopic dilation before the start of the study (3/13, 23.1%) and those who had not (2/7, 28.6%).

### 3.4. Safety analysis

Hypokalemia was detected in 4 of the 20 patients (20.0%) in the EBD/OHA group and none of the patients in the EBD alone group (Table 3). The hypokalemia in the 4 patients in the EBD/OHA group was considered mild (serum potassium level of 3.0–
3.5 mmol/L) in 1 case (5.0%), moderate (serum potassium level of 2.5–3.0 mmol/L) in 2 cases (10.0%), and severe (serum potassium level < 2.5 mmol/L) in 1 case (5.0%). The hypokalemia was improved in all 4 patients after active potassium supplementation. In addition, 1 patient (5.0%) in the EBD/OHA group developed symptoms of apathy and somnolence that resolved gradually after 4 days of drug withdrawal. No other complications, such as hypertension and hyperglycemia, occurred in either group during the follow-up period.

4. Discussion

The main findings of this study were that oral administration of a steroid/gel mixture for 8 weeks after EBD resulted in significant reductions in the restenosis rate, degree of restenosis, and severity of dysphagia symptoms. In addition, there was a trend toward a decrease in the number of additional EBD sessions needed during the 3-month follow-up, although this was not a statistically significant effect. Taken together, our data indicate that the oral administration of steroid/gel mixture after EBD might be a promising method of reducing the incidence of restenosis, with lower risks of perforation and easier operation than injection of steroids.

Previous studies conducted outside of China, particularly those in western countries, have reported that chronic acid exposure (ie, GERD) can underlie up to 80% of all cases of esophageal stenosis. However, ESD and surgery are more common in western countries, have reported that chronic acid exposure (ie, GERD) can underlie up to 80% of all cases of esophageal stenosis. Although some investigators have reported that EBD is effective at treating benign esophageal stricture, with lower risks of perforation and easier operation than injection of steroids, our analysis or their interpretation.

In this study, the restenosis rate was significantly lower in the EBD/OHA group than in the EBD alone group. Furthermore, the dysphagia symptom grade during follow-up and the restenosis severity were both lower in the EBD/OHA group than in the EBD alone group. These results suggest that oral administration of the steroid/gel mixture was effective at preventing post-EBD restenosis. Although the number of additional EBD sessions did not differ significantly between groups, a clear trend was evident. Notably, 3 of the patients with restenosis in the EBD alone group were lost to follow-up before repeat EBD was performed, so it is possible that these missing data may have skewed the analysis and underestimated the benefits of the steroid/gel mixture on the number of additional EBD treatments needed.

No significant differences were found between the 2 groups in age, sex, stricture etiology, stricture location, diameter of the narrowest segment, or esophageal diameter post-EBD, suggesting that the baseline characteristics of the 2 groups were similar. However, 1 baseline parameter that did differ between groups was the past history of dilation therapy, with 35% of patients in the EBD/OHA group having received previous dilation treatment compared with none in the EBD alone group. However, the restenosis rate did not differ significantly between patients in the EBD/OHA group who had received endoscopic dilation before the study start and those who had not. Thus, we do not consider this difference between groups to have impacted on the results of our analysis or their interpretation.

Our study has several limitations. First, this was a retrospective analysis and so may be prone to selection and information bias. Second, this was a single-center study with limited sample size, so the generalizability of the findings is unknown. Furthermore, the small sample size may have resulted in the analysis being underpowered to detect some real differences (for example, number of additional EBD sessions). Third, the follow-up period was only 3 months because most patients were followed only for this duration, so the longer-term effects of the steroid/gel mixture remain unknown. Finally, the study period is long, but it corresponds to the period during which we used the techniques described in our study. It is true that some preoperative, perioperative, and postoperative management methods might have evolved during the study period, possibly affecting the results. A randomized controlled trial with a larger sample size will be needed to confirm our findings.

The oral administration of steroid/gel mixture after EBD might be a promising technique for reducing the incidence of restenosis, with lower risks of perforation and easier operation than injection of steroids. However, further evaluations and confirmation in prospective studies with larger sample sizes are needed before this treatment method is applied more widely in the clinical setting.
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