

Complete Esophageal Obstruction Following Endoscopic Variceal Ligation

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Endoscopic variceal ligation (EVL) is the standard-of-care therapy for treating and preventing recurrence of acute esophageal variceal hemorrhage.¹ EVL can also be used to prevent a patient's first variceal bleeding episode, particularly in patients who have medium or large varices showing high-risk signs for bleeding or patients who are intolerant to β -blocker therapy.¹⁻³ EVL has supplanted the use of endoscopic sclerotherapy (EST), as EVL has a lower overall complication rate and equal or better efficacy for controlling acute bleeding and lowering rebleeding rates.⁴⁻¹¹ Complications of EST include stricture formation, ulceration of esophageal mucosa, rebleeding, hematoma formation, perforation, spontaneous bacterial peritonitis, and pulmonary infections.^{3,6-9,11,12} Complications of EVL, on the other hand, are generally benign.^{4,6-9,11} However, dysphagia following EVL has often been reported in the literature.^{6,10,11,13} Complete esophageal obstruction causing dysphagia has been reported only once previously in the literature.¹⁴ We report the second case of complete esophageal obstruction following EVL. Our patient was managed conservatively and experienced a good outcome.

Case Report

A 67-year-old woman with Child-Pugh class A cirrhosis secondary to primary biliary cirrhosis (PBC) presented to our endoscopy suite to undergo her second esophago-gastroduodenoscopy (EGD) for band ligation of known esophageal varices. The patient had been diagnosed with PBC 3 years previously. Four months prior to presenta-

tion, she had been referred to our institution for consideration of orthotopic liver transplantation. At that time, she had never been screened for esophageal varices. An EGD performed shortly thereafter at an outside institution detected grade 2-3 varices. Treatment with a nonselective β -blocker was initiated; however, the patient was intolerant to this drug due to her history of asthma. Therefore, she began a series of EVLs for primary prophylaxis against variceal hemorrhage. These procedures revealed 4 columns of grade 3 varices with high-risk signs for bleeding (ie, red wales and cherry red spots). During her first EVL procedure, a total of 5 bands were successfully placed without adverse outcomes.

At the time of the patient's second EGD for EVL, grade 3 varices were seen and again showed high-risk features. Three bands were successfully placed in the distal esophagus. In the recovery suite following the procedure, the patient complained of severe chest pain (giving it a score of 10 on a scale from 1 to 10) and was unresponsive to both intravenous meperidine and a solution of aluminum hydroxide, magnesium hydroxide, simethicone, and viscous lidocaine. Within minutes of drinking the solution, the patient vomited white liquid with no evidence of blood. She was admitted to our hospital for further evaluation and treatment.

Upon admission to our hospital, the patient was afebrile with normal vital signs (a heart rate of 60 beats per minute, respiratory rate of 18 breaths per minute, blood pressure of 110/56 mmHg, and oxygen saturation rate of 100% on room air). Physical examination revealed an elderly female in no acute distress, and cardiopulmonary examination was unremarkable. Abdominal examination was notable for mild epigastric tenderness to palpation but no rebound or guarding. The patient's bowel sounds were normoactive, and the remainder of her examination was normal. Results from laboratory tests taken upon

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Figure 1. A gastrografin swallow study revealing complete obstruction at the level of the distal esophagus. No contrast or air is seen within the stomach.

admission were normal, except for slightly elevated levels of total bilirubin, alkaline phosphatase, and aspartate aminotransferase, all of which were stable. An acute abdominal series was within normal limits.

On the second day of hospitalization, the patient's pain was slightly better; however, she was still unable to tolerate liquids. She was also expectorating all of her oral secretions into an emesis basin and had slight odynophagia. A gastrografin swallow study was performed, showing complete obstruction at the level of the distal esophagus (Figure 1). No contrast or air was noted in the stomach. Due to the obstruction, the patient was started on partial parenteral nutrition.

Fortunately, on the seventh day after her EGD, the patient began to tolerate liquids. She was subsequently discharged the following day.

Discussion

EVL was first introduced in 1986.⁴ Prior to EVL's inception, EST was used to control active variceal bleeding and prevent recurrent hemorrhage. However, due to its induction of tissue injury, EST is associated with complications in nearly 40% of patients.⁴⁻⁶ Complications of EST include stricture formation, ulceration of esophageal mucosa, rebleeding, hematoma formation, perforation, spontaneous bacterial peritonitis, and pulmonary infections.^{4,6-9,11,12} Stricture formation rates are as high as 33%, and dysphagia occurs in 7–30% of patients who undergo EST.^{9,11,12}

EVL has consistently demonstrated equal or better efficacy compared to EST in terms of controlling acute bleeding varices and lowering rebleeding rates and mortality.^{4,7,8,9,11} As these benefits have been achieved with fewer complications, EVL has become the standard-of-care treatment for esophageal varices.^{1,4-10} To perform EVL, the clinician places a small, elastic O-ring over a small area of esophageal mucosa and submucosa.^{4,7,15} The ensnared tissue is strangulated, leading to ischemia and, eventually, sloughing, fibrosis, and variceal obliteration.^{7,15} Given that this technique is purely mechanical, transmural inflammation is not invoked, and systemic complications are not seen.^{4,10} Complications commonly described after EVL include stricture formation, ulcers, ulcer bleeding, pneumonia, and spontaneous bacterial peritonitis.^{4,6-9,11} Ulcer formation is nearly universal, documented in 94% of patients on follow-up EGD.^{9,11,15} Stricture formation occurs in 0% of patients in some reported series (n=64 and n=38), and an early meta-analysis comparing EVL and EST revealed a stricture odds ratio of 0.10 in favor of EVL.^{4,7,9} The occurrence of transient dysphagia (lasting 24–72 hours) is variable; this complication has been reported in anywhere from 0% to 75% of patients in various published case series.^{11,13} Engorged banded varices are the presumed cause of this phenomenon.⁶ Another unique complication described by Berner and associates is altered lower esophageal sphincter relaxation following EVL.¹⁰ However, significant differences in acid reflux and esophageal motility were not demonstrated.¹⁰

To date, there has been only 1 case of esophageal obstruction as a complication of EVL that has been reported in the literature.¹⁴ A 58-year-old man with cirrhosis secondary to hepatitis C virus infection who had a history of esophageal variceal bleeding had undergone 2 sessions of EST. He subsequently underwent 2 sessions of EVL 3 weeks apart. At the time of the first EVL session, grade 3 varices were noted, and 4 bands were placed without difficulty. The second EVL session again revealed grade 3 varices, for which an additional 4 bands were placed without complications. After endoscopy, the patient resumed a normal diet and experienced chest discomfort and sialorrhea. An emergent upper endoscopy revealed food stuck in the esophagus above the banded varices. On inspection, the newly banded varices had completely obstructed the esophageal lumen. The authors postulated that the newly banded varices had swollen, thereby completely occluding the lumen.

Three pathophysiologic mechanisms have been proposed to explain the occurrence of dysphagia following EVL. The most commonly proposed mechanism is stricture formation, which typically presents as late dysphagia, allowing time for fibrosis progression and stricture formation.^{6,7,13,15} Dysphagia that presents soon after EVL may be secondary to transient alterations in esophageal motility.^{10,13,15} Finally, as in this case report, complete esophageal obstruction can occur.¹⁴

We postulate that complete obstruction of the esophagus is an exceedingly rare complication of EVL that occurs due to several interrelated factors, not simply engorged varices that fill the esophageal lumen. Clearly, the size of the banded varix contributes to this phenomenon. However, the size of the banding cap limits the volume of each individual varix that can be banded.¹⁵ Therefore, the size of the banded varix cannot be the only factor; otherwise, this complication would be more common. In this case report and in the first reported case of complete esophageal obstruction due to EVL, both patients underwent 2 EVL sessions; in the first case, the patient also underwent 2 prior EST sessions. Prior to undergoing these procedures, the esophageal mucosa may already have abnormalities, such as undetected strictures from prior therapy or an unrelated Schatzki ring. If a varix is banded proximally to 1 of these areas, it is possible that the peristaltic action of the esophagus could propagate the banded varix into the strictured segment, creating a ball-valve effect. The effects of this scenario would be similar to those resulting from a food impaction caused by a Schatzki ring. The stalk created by a banded varix would prevent further distal propagation of the varix until it sloughs off 1–2 weeks after placement of the band.

Summary

Complete esophageal obstruction has been described in the literature only once before. In this case report, we present the second such case. Immediate complaints of dysphagia after banding—along with the presence of sialorrhea or the inability to tolerate liquids—should suggest the possibility of obstruction. Obstruction can be documented by a contrast swallow study. If the patient has not eaten recently (ie, there is no chance of a food impaction), we do not recommend performing a repeat endoscopy, as it could dislodge a band and cause bleeding. Conservative measures such as intravenous fluids and parenteral nutrition may be needed. With time, the varix should slough, and dysphagia should be relieved.

References

- Garcia-Tsao G, Sanyal AJ, Grace ND, Carey W; Practice Guidelines Committee of the American Association for the Study of Liver Diseases; Practice Parameters Committee of the American College of Gastroenterology. Prevention and management of gastroesophageal varices and variceal hemorrhage in cirrhosis. *Hepatology*. 2007;46:922-938.
- Schepke M, Kleber G, Nürnberg D, et al. Ligation versus propranolol for the primary prophylaxis of variceal bleeding in cirrhosis. *Hepatology*. 2004;40:65-72.
- Lay CS, Tsai YT, Lee FY, et al. Endoscopic variceal ligation versus propranolol in prophylaxis of first variceal bleeding in patients with cirrhosis. *J Gastroenterol Hepatol*. 2006;21:413-419.
- Stiegmann GV, Goff JS, Michaletz-Onody PA, et al. Endoscopic sclerotherapy as compared with endoscopic ligation for bleeding esophageal varices. *N Engl J Med*. 1992;326:1527-1532.
- Sivak MV Jr, Catalano MF. Endoscopic ligation of esophageal varices. *Ann Intern Med*. 1993;119:87-88.
- Schmitz RJ, Sharma P, Badr AS, Qamar MT, Weston AP. Incidence and management of esophageal stricture formation, ulcer bleeding, perforation, and massive hematoma formation from sclerotherapy versus band ligation. *Am J Gastroenterol*. 2001;96:437-441.
- Laine L, Cook D. Endoscopic ligation compared with sclerotherapy for treatment of esophageal variceal bleeding. A meta-analysis. *Ann Intern Med*. 1995;123:280-287.
- Al Traif I, Fachartz FS, Al Jumah A, et al. Randomized trial of ligation versus combined ligation and sclerotherapy for bleeding esophageal varices. *Gastrointest Endosc*. 1999;50:1-6.
- Laine L, el-Newihi HM, Migikovsky B, Sloane R, Garcia F. Endoscopic ligation compared with sclerotherapy for the treatment of bleeding esophageal varices. *Ann Intern Med*. 1993;119:1-7.
- Berner JS, Gaing AA, Sharma R, Almenoff PL, Muhlfelder T, Korsten MA. Sequelae after esophageal variceal ligation and sclerotherapy: a prospective randomized study. *Am J Gastroenterol*. 1994;89:852-858.
- Saeed ZA, Stiegmann GV, Ramirez FC, et al. Endoscopic variceal ligation is superior to combined ligation and sclerotherapy for esophageal varices: a multicenter prospective randomized trial. *Hepatology*. 1997;25:71-74.
- Svoboda P, Kantorová I, Ochmann J, Kozumplík L, Marsová J. A prospective randomized controlled trial of sclerotherapy vs ligation in the prophylactic treatment of high-risk esophageal varices. *Surg Endosc*. 1999;13:580-584.
- Goff JS, Reveille RM, Van Stiegmann G. Endoscopic sclerotherapy versus endoscopic variceal ligation: esophageal symptoms, complications, and motility. *Am J Gastroenterol*. 1988;83:1240-1244.
- Saltzman JR, Arora S. Complications of esophageal variceal band ligation. *Gastrointest Endosc*. 1993;39:185-186.
- Holderman WH, Etzkorn KP, Patel SA, Harig JM, Watkins JL. Endoscopic findings and overtube-related complications associated with esophageal variceal ligation. *J Clin Gastroenterol*. 1995;21:91-94.

Review

Primary Prophylaxis of Variceal Bleeding

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Variceal hemorrhage is the most serious and dreaded complication of portal hypertension, 1 of the consequences of liver cirrhosis. Esophageal varices, which are usually caused by portal hypertension, have an annual incidence rate of approximately 5–10% in patients with cirrhosis. Small varices may increase in size, developing into large varices at a rate of 5–30% per year.^{1,2} Due to the risk of bleeding, primary prevention of hemorrhage is needed in patients with esophageal varices. Current options for primary prophylaxis include nonselective β -blocker (NSBB) therapy and endoscopic variceal ligation (EVL).

The patient presented by Nikoloff and colleagues exemplifies a very commonly encountered situation in daily clinical practice in hepatology units: the need for primary prophylaxis for bleeding caused by esophageal varices.³ This patient first received NSBB therapy despite having a history of asthma (which is considered to be a contraindication to NSBB therapy in many centers, although this therapy is permitted under close monitoring in some patients with asthma). However, the patient had to discontinue this treatment due to worsening breathing problems. As the patient had high-risk varices that were detected via endoscopy, EVL was used as an alternative option for the prevention of bleeding. The complication that occurred following the banding procedure is interesting but extremely rare.

The decision to use NSBBs despite a commonly considered contraindication in this patient may have

been based upon the easy administration, low expense, and relatively few adverse effects of these agents, which, importantly, are usually reversible after treatment discontinuation. NSBBs are effective; in the 9 randomized trials conducted to date, NSBB therapy reduced both bleeding and mortality rates, with the latter difference being at the threshold of statistical significance.^{4–12} In addition, NSBBs protect against gastric mucosal bleeding from portal hypertensive gastropathy, which causes a proportion of first bleeding episodes. It is also cheaper to use NSBBs than EVL. According to the most recent meta-analysis of 16 randomized trials comparing NSBBs with EVL, the number of patients who need to be treated with EVL to prevent 1 bleeding episode is 11. Considering that the average number of endoscopic sessions required to eradicate varices is 3, at least 33 endoscopic procedures are necessary to prevent 1 bleeding episode as compared with NSBBs. Using EVL rather than NSBBs would yield no benefit in reducing mortality and would entail much greater expense, although there is a 9% reduction in first bleeding.¹³

EVL is considered to be a relatively safe technique. As with any therapy, the risk-to-benefit ratio should be considered for different situations. In the patient treated by Nikoloff and associates, there was no therapeutic alternative to EVL.³ It should be noted that while the risk for the first bleeding episode is approximately 20% within 1–2 years, the risk of rebleeding is 70% within the same time period; thus, the risk of EVL complications must be interpreted in this light, as complications have a higher impact on primary prophylaxis when patients are asymptomatic. The most important predictors of hemorrhage are the size of the varices, the presence of red signs on the varices, and the patient's Child-Pugh class.^{1,14} Although the patient treated by Nikoloff and coworkers had only Child-Pugh class A cirrhosis, the patient had grade 3 varices with red wales and cherry red spots (ie, large varices with high-risk signs).³ Therefore, the need for primary prophylaxis with EVL was clear due to the patient's intolerance of NSBB therapy because of her asthma.

EVL is a relatively simple procedure: First, a diagnostic upper endoscopy is performed to identify which varices need to be treated. Elastic bands are placed on the varices from just above the gastroesophageal junction, ascending proximally in a spiral fashion in order to avoid occlusion of the lumen; while partial occlusion cannot be avoided, it is usually asymptomatic or causes only transient dysphagia. During the initial EVL session, 3–8 bands are commonly used, although there is no set limit to the number of bands that can be used. However, a randomized study showed that the use of more than 6 bands per session did not result in better outcomes; in fact, it prolonged procedural time and increased the number of

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misfired bands, though the number of complications did not increase.¹⁵ Endoscopic follow-up is needed to assess the eradication of varices; if they are still detected, additional bands can be applied. In the patient managed by Nikoloff and colleagues, the placement technique was followed as described above, and the patient was scheduled for a second EVL session, as she had had no problems with her first session.³ However, the time interval between the 2 banding sessions is not clear. Guidelines from the American Association for the Study of Liver Diseases recommend that EVL sessions be repeated every 1–2 weeks until variceal obliteration is achieved, with the first surveillance esophagogastroduodenoscopy performed 1–3 months after variceal obliteration and every 6–12 months thereafter to check for variceal recurrence.¹⁴

A universal complication of EVL is mucosal ulceration at the ligation sites due to tissue necrosis produced by the band. This is usually asymptomatic, although it sometimes causes bleeding. The ulcers usually heal within 14 days.

Because the ulcers are superficial, the development of strictures after EVL is rare, occurring at a rate of 0–1%.^{16,17} In an isolated case, a stricture was reported following inadvertent binding of 2 varices with a single band at the same level in opposite walls.¹⁸ Local necrosis and fatal perforation of the esophagus can occur, particularly in patients taking corticosteroids.¹⁹ Although pain is uncommon, some patients experience pain immediately after the bands are placed, and dysphagia and odynophagia may occur. Esophageal spasm may be responsible for these symptoms. Transient bacteremia may occur, although this condition is thought to be of no clinical significance; therefore, routine antibiotic prophylaxis is not needed.²⁰ Pulmonary infections are uncommon.^{21,22} Unusual complications—such as esophageal obstruction, paraplegia, mesenteric vein thrombosis, banding of laryngeal mucosa, or pyogenic meningitis—have also been reported in the literature, usually in single case reports.^{23–28}

Esophageal obstruction, which was documented in the case reported by Nikoloff and associates, is an extremely rare complication, and the authors identified 1 other such case in the literature.³ We have identified an additional case in the literature: a 65-year-old woman with cirrhosis who had EVL for secondary prophylaxis of variceal bleeding and developed total esophageal obstruction 1 day after EVL.²⁸ Her previous EVL session had been 2 weeks earlier. The obstruction was caused by a necrotic and obstructing mass involving 3 bands. The patient received conservative treatment, and after 2 weeks, the obstruction completely resolved. Nikoloff and colleagues suggest that the obstruction in their patient occurred as a result of luminal compromise secondary to tissue edema and necrosis at the banding site.³ They recommend spiral

application of the bands—which is the recommended technique, particularly in patients who need multiple sessions—so as to avoid developing a “transverse” that could potentially cause luminal obstruction.

As only 3 cases of esophageal obstruction after EVL have been published in the literature and the complication is so dramatic, it can be assumed that esophageal obstruction must be extremely rare. It is also reasonable to assume that placing a band close to mucosa that is already damaged with edema, inflammation, and/or necrosis from previous endoscopic procedures may cause further injury and edema. Nikoloff and coworkers also postulate that a previously unnoticed esophageal abnormality, such as a Schatzki ring, could have acted as a trigger in their patient.³

Regardless of the reason for the obstruction, it would be sensible to ensure that bands are applied in a spiral fashion, as is commonly recommended. It is clear from these 3 cases that treatment for obstruction following EVL should be conservative, as this type of therapy was successful in each case. It is reasonable to expect that further endoscopic interventions could result in complications such as bleeding or perforation.

Summary

The first choice for primary prophylaxis of esophageal variceal bleeding is an NSBB, such as propranolol, as these agents are cheaper than EVL and easy to administer. Carvedilol could be an alternative option.²⁹ NSBBs are effective for controlling variceal hemorrhage and bleeding from gastric mucosa, as well as providing potential benefits related to the reduction of bacterial translocation and infections.³⁰ EVL should be offered to patients who are unable to use NSBBs, as in the case presented by Nikoloff and coworkers, or patients in whom drug therapy has failed.^{3,31} Patient preference should also be taken into account whenever possible. EVL is a relatively safe technique for primary prophylaxis of esophageal varices, with a low rate of complications (although serious complications may occur, as in the reported case).

References

1. North Italian Endoscopic Club for the Study and Treatment of Esophageal Varices. Prediction of the first variceal hemorrhage in patients with cirrhosis of the liver and esophageal varices. A prospective multicenter study. *N Engl J Med.* 1988;319:983-989.
2. Chalasani N, Boyer TD. Primary prophylaxis against variceal bleeding: beta-blockers, endoscopic ligation, or both? *Am J Gastroenterol.* 2005;100:805-807.
3. Nikoloff MA, Riley TR 3rd, Schreibman IR. Complete esophageal obstruction following endoscopic variceal ligation. *Gastroenterol Hepatol (N Y).* 2011;7:557-559.
4. Propranolol prevents first gastrointestinal bleeding in non-ascitic cirrhotic patients. Final report of a multicenter randomized trial. The Italian Multicenter Project for Propranolol in Prevention of Bleeding. *J Hepatol.* 1989;9:75-83.

5. Prophylaxis of first hemorrhage from esophageal varices by sclerotherapy, propranolol or both in cirrhotic patients: a randomized multicenter trial. The PROVA Study Group. *Hepatology*. 1991;14:1016-1024.
6. Andreani T, Poupon RE, Balkau BJ, et al. Preventive therapy of first gastrointestinal bleeding in patients with cirrhosis: results of a controlled trial comparing propranolol, endoscopic sclerotherapy and placebo. *Hepatology*. 1990;12:1413-1419.
7. Conn HO, Grace ND, Bosch J, et al. Propranolol in the prevention of the first hemorrhage from esophagogastric varices: a multicenter, randomized clinical trial. The Boston-New Haven-Barcelona Portal Hypertension Study Group. *Hepatology*. 1991;13:902-912.
8. Pascal JB, Cales P. Propranolol in the prevention of first upper gastrointestinal tract hemorrhage in patients with cirrhosis of the liver and esophageal varices. *N Engl J Med*. 1987;317:856-861.
9. Colman J, Jones P, Finch C, Dudley F. Propranolol in the prevention of variceal hemorrhage in alcoholic cirrhotic patients [abstract]. *Hepatology*. 1990;12:851.
10. Strauss E, Desa M, Albano CM, et al. A randomized controlled trial for the prevention of the 1st upper gastrointestinal bleeding due to portal hypertension in cirrhosis—sclerotherapy or propranolol versus control groups [abstract]. *Hepatology*. 1988;8:1395.
11. Idéo G, Bellati G, Fesce E, Grimoldi D. Nadolol can prevent the first gastrointestinal bleeding in cirrhotics: a prospective, randomized study. *Hepatology*. 1988;8:6-9.
12. Lebre C, Poynard T, Capron JP, et al. Nadolol for prophylaxis of gastrointestinal bleeding in patients with cirrhosis. A randomized trial. *J Hepatol*. 1988;7:118-125.
13. Burroughs AK, Tsochatzis EA, Triantos C. Primary prevention of variceal haemorrhage: a pharmacological approach. *J Hepatol*. 2010;52:946-948.
14. Garcia-Tsao G, Sanyal AJ, Grace ND, Carey W. Prevention and management of gastroesophageal varices and variceal hemorrhage in cirrhosis. *Hepatology*. 2007;46:922-938.
15. Ramirez FC, Colon VJ, Landan D, Grade AJ, Evanich E. The effects of the number of rubber bands placed at each endoscopic session upon variceal outcomes: a prospective, randomized study. *Am J Gastroenterol*. 2007;102:1372-1376.
16. Goff JS, Reveille RM, Stiegmann GV. Three years experience with endoscopic variceal ligation for treatment of bleeding varices. *Endoscopy*. 1992;24:401-404.
17. Stiegmann GV, Goff JS, Michaletz-Onody PA, et al. Endoscopic sclerotherapy as compared with endoscopic ligation for bleeding esophageal varices. *N Engl J Med*. 1992;326:1527-1532.
18. Rai RR, Nijhawan S, Singh G. Post-ligation stricture: a rare complication. *Endoscopy*. 1996;28:406.
19. Schoonbroodt D, Zipf A, Jung M. Local necrosis and fatal perforation of oesophagus after endoscopic ligation. *Lancet*. 1994;344:1365.
20. Maulaz EB, de Mattos AA, Pereira-Lima J, Dietz J. Bacteremia in cirrhotic patients submitted to endoscopic band ligation of esophageal varices. *Arq Gastroenterol*. 2003;40:166-172.
21. Berner JS, Gaing AA, Sharma R, Almenoff PL, Muhlfelder T, Korsten MA. Sequelae after esophageal variceal ligation and sclerotherapy: a prospective randomized study. *Am J Gastroenterol*. 1994;89:852-858.
22. Laine L, Cook D. Endoscopic ligation compared with sclerotherapy for treatment of esophageal variceal bleeding. A meta-analysis. *Ann Intern Med*. 1995;123:280-287.
23. Debette S, Gauvrit JY, Desseaux G, et al. Paraplegia after ligation of esophageal varices. *Neurology*. 2003;60:879-880.
24. Malpani NK, Somani AK, Vardhan V, Goyal VP. Inadvertent banding of laryngeal mucosa during endoscopic variceal band ligation. *Indian J Gastroenterol*. 1997;16:121.
25. Nagamine N, Kaneko Y, Kumakura Y, Ogawa Y, Ido K, Kimura K. Occurrence of pyogenic meningitis during the course of endoscopic variceal ligation therapy. *Gastrointest Endosc*. 1999;49:110-113.
26. Tachibana I, Yoshikawa I, Sano Y, Tabaru A, Murata I, Otsuki M. A case of mesenteric venous thrombosis after endoscopic variceal band ligation. *J Gastroenterol*. 1995;30:254-257.
27. Saltzman JR, Arora S. Complications of esophageal variceal band ligation. *Gastrointest Endosc*. 1993;39:185-186.
28. Verma D, Pham C, Madan A. Complete esophageal obstruction: an unusual complication of esophageal variceal ligation. *Endoscopy*. 2009;41(suppl 2):E200-E201.
29. Tripathi D, Ferguson JW, Kochar N, et al. Randomized controlled trial of carvedilol versus variceal band ligation for the prevention of the first variceal bleed. *Hepatology*. 2009;50:825-833.
30. Thalheimer U, Bosch J, Burroughs AK. How to prevent varices from bleeding: shades of grey—the case for nonselective beta blockers. *Gastroenterology*. 2007;133:2029-2036.
31. Thalheimer U, Triantos C, Goulis J, Burroughs AK. Management of varices in cirrhosis. *Expert Opin Pharmacother*. 2011;12:721-735.