A Case of Sengstaken-Blakemore Tube-Induced Esophageal Rupture Repaired by Endoscopic Clipping

Jin Hwan Jung, Jin Il Kim, Jun Ho Song, Jeong Ho Kim, Sang Hun Lee,
Dae Young Cheung, Soo Heon Park and Jae Kwang Kim

Abstract

A 57-year-old man was admitted to another hospital for hematemesis due to heavy drinking. A Sengstaken-Blakemore tube was inserted and the patient was transferred to our hospital. The patient's ensuing movements inadvertently caused an esophageal rupture 2.5 cm in size. Since the patient's condition was stable, treatment via endoscopic repair using metallic clips was chosen over emergency surgery. Two hemo-clips were fixed at the ends of the ruptured area; by employing an endoscopic detachable snare, the ruptured area was carefully repaired with 10 metallic clips. As a result, the esophageal rupture could be successfully repaired by endoscopic procedure rather than performing surgery.

Key words: Sengstaken-Blakemore tube, esophageal rupture, Boerhaave's syndrome, endoscopic clipping

(Intern Med 50: 1941-1945, 2011)

(DOI: 10.2169/internalmedicine.50.5432)

Introduction

Although esophageal rupture occurs rarely, because of the anatomical characteristic of lacking serosa, inflammation easily spreads, leading to high complication rates and mortality. There are many causes for such rupture, including primarily artificial manipulation such as insertion of an esophageal tube, endoscopic examination, and esophageal balloon dilatation (1).

The protocol for treatment is to perform surgical drainage followed by primary repair and reconstruction. Recently, for some select patients, conservative management or treatment other than surgery such as endoscopic repair has been attempted (2, 3). In the previous cases where treatment by endoscopic repair was successful, the size of the rupture was generally smaller than 10 mm. We herein document a relatively large rupture of 2.5 cm that was effectively treated by endoscopic clipping.

The Sengstaken-Blakemore tube is used for emergency hemostasis in esophageal variceal bleeding, and although rare, it can cause esophageal rupture, which is a complication that may be fatal (4). In the present case, the rupture was caused by fixation of the Sengstaken-Blakemore tube to

the bed railing, warranting extra attention.

We herein report a case of an esophageal rupture in Mallory-Weiss syndrome that resulted from the insertion and fixation of a Sengstaken-Blakemore tube, which was repaired with endoscopic metallic clipping.

Case Report

A 57-year-old male patient was transferred from another hospital complaining with 300 mL of hematemesis preceded by heavy alcohol consumption. In the previous hospital, esophagogastroduodenoscopy was performed to achieve hemostasis. However, the exact bleeding site could not be found due to profuse bleeding. Neither sclerotherapy nor an injection method was performed. Therefore, under the suspicion of esophageal variceal bleeding, the patient was transferred to our hospital after insertion of a Sengstaken-Blakemore tube and intubation of an endotracheal tube; 300 mL of air was injected to the gastric balloon of the Sengstaken-Blakemore tube, and the esophageal balloon was maintained at a pressure of 40 mmHg. For transfer, the end of the tube was fixed to the patient's bed railings. As for pertinent history, the patient was diagnosed with alcoholic liver disease 4 years ago but had not received treatment, and

Figure 1. (A) Simple chest X-ray findings. The esophageal balloon of Sengstaken-Blakemore tube was located in the upper esophagus, and the gastric balloon was not visible (arrow). (B) Gastroduodenoscopy findings. On initial endoscopic examination, there was a 2.5×1.5 cm sized ulcer at the lower esophagus and a 1.0×0.6 cm sized ulcer at the esophagogastric junction. (C) Gastroduodenoscopy findings. With cap attached to the endoscope, it shows a 2.5×1.5 cm sized ulcer with exudates at the ulcer base.

Figure 2. Chest CT findings. (A) There is multifocal free air and adjacent fistulous tract at the lower esophagus (arrow). (B) Free air, edema in the lower esophagus wall and left pleural effusion in the left lower esophagus was still noted (arrow). (C) Remained edematous wall thickening and multiple metallic clips at the lower esophagus is noted (arrow). There is no mediastinal free air.

had been consuming 240 g of alcohol daily for the past 20 years.

At the time of admission to our hospital, the patient's mental status was alert. Vital signs were as follows: Blood pressure was 132/104 mmHg, pulse rate was 109/minute, respiration rate was 22/minute, and body temperature was 37.8°C. Upon physical examination, the conjunctivae were pale, and icteric sclerae were not detected. There was no presence of ascites, hepatomegaly, or splenomegaly, but epigastric tenderness was noted while rebound tenderness was not. In the complete blood count, the measured hemoglobin level was 9.5 g/dL, white blood cell count was 4,440/mm³, and platelet count was 31,000/mm³. In the blood chemistry test, the measured level of AST was 241 IU/L, ALT was 89 IU/L, total bilirubin was 1.19 mg/dL, ALP was 131 IU/L, γ-GTP was 119 IU/L, prothrombin time was INR 1.02, albumin was 3.24 g/dL, BUN was 36.1 mg/dL, and creatinine was 1.36 mg/dL. The simple chest and abdominal X-ray showed nonspecific findings, aside from the endotracheal tube, the esophageal balloon and gastric balloon of the Sengstaken-Blakemore tube. On the second day following admission, his vital signs were stabilized. In the follow-up

simple chest X-ray, the esophageal balloon of the Sengstaken-Blakemore tube was located in the upper esophagus, and the gastric balloon was not visible (Fig. 1A). Subsequently, the Sengstaken-Blakemore tube was removed. After removal of the Sengstaken-Blakemore tube, we tried to put air in the gastric balloon, but the air leaked from the balloon. Therefore, we could confirm the rupture of the gastric balloon of the Sengstaken-Blakemore tube. An esophagogastrroduodenoscopy was performed, and an ulcerous lesion (2.5×1.5 cm) with blood clots was observed in the lower esophagus, suggestive of a rupture caused by the Sengstaken-Blakemore tube (Fig. 1B, 1C). The chest CT revealed the presence of free air and a fistulous tract on the left side of the lower esophagus (Fig. 2A), allowing us to approach the diagnosis of esophageal rupture. Hence, antibiotics were administrated and continued non per os was maintained.

Since the general condition of the patient was relatively stable, the decision was made to maintain conservative management and to consider surgery only in the case of a turn for the worse. The patient maintained non per os, and in the chest CT taken on the 10th day of admission, free air,

Figure 3. Gastroduodenoscopy findings. (A) Argon plasma coagulation was applied to the mucosal defect. (B) Two metallic clips were applied to either side of the ulcer margin. (C) Ulcer margins were approximated by detachable snare. (D) The ulcer opening was successfully closed by endoscopic application of metallic clips.

edema in the lower esophageal wall and left pleural effusion in the left lower esophagus was still noted (Fig. 2B). On the 12th day of admission, a follow-up esophagogastroduodenoscopy was performed, and an esophageal rupture in the lower esophagus (2.5×1.5 cm) was detected. For optimal repair, argon plasma coagulation was performed to induce inflammation, and the rupture area was approximated using 2 endoscopic hemoclips (Olympus, EZ-CLIP, long type, 90°) and a detachable snare. Afterward, the rupture area was repaired with 10 endoscopic hemoclips (Fig. 3). In the follow-up esophagogastroduodenoscopy performed 11 days later, the endoscopic hemoclips were well positioned in the repaired area, and leakage in the esophagus was not observed on the chest CT (Fig. 2C) and esophagography (Fig. 4A). The findings continued to be nonspecific later on, and diet was eventually initiated. In the follow-up esophagogastroduodenoscopy performed 2 months later at outpatient clinic, the rupture area was well repaired (Fig. 4B). Presently, the patient is under follow-up observation at our outpatient clinic.

Discussion

An esophageal rupture occurs rarely, but since it can be fatal once developed, an early diagnosis and decision for the appropriate treatment strategy is important.

A simple chest X-ray, chest CT, esophagography, and esophagogastroduodenoscopy may be used to diagnose an esophageal rupture. Chest CT is effective in the detection of air, abscess, and the air fluid level in tissues around the esophagus, and is also useful for a diagnosis in the asymptomatic cases. As a means to directly examine the rupture area, esophagogastroduodenoscopy is suitable for follow-up observation (5). The esophagography may be used to aid the assessment of the rupture site, but the false negative rate runs as high as 10 % (6). Therefore, it is best to perform both a chest CT as well as an esophagogastroduodenoscopy.

For treatment, surgery is performed in most patients, and drainage of the inflammation area and repair of the rupture are also performed. Pneumothorax, pneumoperitoneum, sepsis, heart failure, pulmonary failure, and shock are indications for emergency operation (7). Nevertheless, when the rupture is limited to the vicinity of the mediastinum, with

Figure 4. (A) Esophagography findings. Multiple metallic clips at the lower esophagus is shown 11 days after endoscopic clipping (arrow). There is no abnormal leakage of contrast agent. (B) Gastro-duodenoscopy findings. The rupture area is well repaired and remained one hemoclip is shown.

mild symptoms, without an associated pleural inflammation, and without sepsis, conservative management such as non per os, Levin tube insertion, mediastinal drainage, broad spectrum antibiotics, and total parenteral nutrition as well as endoscopic repair may be performed (8, 9). Recently, it has been reported that in patients with a small rupture limited to the mediastinum and without sepsis, conservative management and endoscopic clips or endoscopic stents are helpful (2, 3).

For treatment selection, the location as well as the size of the rupture, the presence or absence of underlying diseases in the esophagus, the time interval period to diagnosis of rupture, the condition of the esophagus, the injury level of adjacent tissues, age, and the general condition are factors to be considered. In cases where esophageal rupture was treated within 24 hours of development, a mortality rate of 10-20% was shown, but after 48 hours, approximately 60% was demonstrated (10). In several reports, cases treated with non-operative management concerned patients with ruptures smaller than 10 mm in size, while surgery was recommended for ruptures larger than 25 mm (11). In the present case, the rupture was limited to the mediastinum and sepsis had not developed. However, the size of the rupture measured 2.5 cm, which is relatively large according to the previous reports. Fortunately, the patient's general condition was fair and thus paired with non per os and antibiotics treatments; endoscopic clipping was a favorable procedure to be attempted. The interval period from esophagus rupture

and endoscopic repair was approximately 10 days. For effective repair, the epithelium formed in the fistulous tract was first removed by inducing inflammation with argon plasma coagulation, and endoscopic clipping was performed subsequently.

Esophageal rupture during the use of the Sengstaken-Blakemore tube rarely occurs, but it is a complication that may be fatal. Therefore, the possibility of such a complication should always be considered, and it is important to assess the location by auscultation or simple chest X-ray considering tube location, pressure, and fixation (12). We herein report a case, in which although the pressure and location of balloons were cautiously monitored, the fixation of the Sengstaken-Blakemore tube to the bed railing delivered excessive force to the esophagus mucosa and consequently caused esophageal rupture. Considering the inadvertent movements of the patient, it may therefore be more appropriate to fix the Sengstaken-Blakemore tube to the helmet, and when the Sengstaken-Blakemore tube is used, sufficient sedation medication of patient is necessary.

In conclusion, we repaired an esophageal rupture, which developed in association with the fixation of the SB tube following insertion, by endoscopic clipping using hemostasis clips.

The authors state that they have no Conflict of Interest (COI).

References

1. Jones WG 2nd, Ginsberg RJ. Esophageal perforation: a continuing challenge. *Ann Thorac Surg* **53**: 534-543, 1992.
2. Shimamoto C, Hirata I, Umegaki E, Katsu K. Closure of an esophageal perforation due to fish bone ingestion by endoscopic clip application. *Gastrointest Endosc* **51**: 736-739, 2000.
3. Abe N, Sugiyama M, Hashimoto Y, et al. Endoscopic nasomediastinal drainage followed by clip application for treatment of delayed esophageal perforation with mediastinitis. *Gastrointest Endosc* **54**: 646-648, 2001.
4. Chong CF. Esophageal rupture due to Sengstaken-Blakemore tube misplacement. *World J Gastroenterol* **11**: 6563-6565, 2005.
5. White CS, Templeton PA, Attar S. Esophageal perforation: CT findings. *Am J Roentgenol* **160**: 767-770, 1993.
6. Sarr MG, Pemberton JH, Payne WS. Management of instrumental perforations of the esophagus. *J Thorac Cardiovasc Surg* **84**: 211-218, 1982.
7. Michel L, Grillo HC, Malt RA. Esophageal perforation. *Ann Thorac Surg* **33**: 203-210, 1982.
8. Janjua KJ. Boerhaave's syndrome. *Postgrad Med J* **73**: 265-270, 1997.
9. Cameron JL, Kieffer RH, Hendrix TR, et al. Selective nonoperative management of contained intrathoracic esophageal disruptions. *Ann Thorac Surg* **27**: 404-408, 1979.
10. Park H, Keum DY, Park HN, Park CK, Lee KS. Clinical analysis and treatment of esophageal perforation. *Korean J Thorac Cardiovasc Surg* **39**: 111-116, 2006.
11. Jang HJ, Kim TH, Lee CM, et al. Two cases of Boerhaave's syndrome treated by endoscopic hemoclippling. *Korean J Gastrointest Endosc* **39**: 359-363, 2009.
12. Seet E, Beevee S, Cheng A, Lim E. The Sengstaken-Blakemore tube: uses and abuses. *Singapore Med J* **49**: e195-e197, 2008.