Esophageal Perforation: A Rare but Fatal Complication of Urgent Sengstaken Blakemore Tube Intubation

Özofagus Perforasyonu: Acil Sengstaken Blakemore Tüp Kullanımı Sonrası Gelişen Nadir Fakat Ölümcül Komplikasyon

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Abstract

Acute variceal bleeding is an important cause of mortality among cirrhotic patients. Endoscopic band ligation and medical vasoactive drugs are the main treatment options for actively bleeding varices. In some cases, these therapies can fail and a Sengstaken Blakemore tube (SBT) is used to stop the bleeding by direct compression of varices. Although it is very effective in stopping variceal bleeding, SBT has some lethal complications. In this paper, we discuss the case of a patient who developed esophageal perforation after insertion of the SBT. (Yoğun Bakım Derg 2013; 4: 28-31)

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Introduction

Approximately 50% of patients with cirrhosis develop esophageal varices, and one third of those patients with esophageal varices bleed. Esophageal varical bleeding is a life threatening complication associated with a mortality that is still in the order of 20% at 6 weeks and, therefore, need urgent intervention (1). Endoscopic treatment with band ligation and pharmacological therapy with vasoactive drugs are the main treatment options for acute variceal bleeding. Unfortunately, in some cases, even a combination of both treatment modalities fails to stop a variceal bleeding. In these situations, a balloon tamponade of SBT as a temporary bridge to other strategies can be used. It stops bleeding by direct compression of varices through inflated esophageal and gastric balloons and succeeds in prevention in up to 90% of cases (2, 3).

Özet

Varis kanaması sirozlu hastalarda önemli bir mortalite nedenidir. Endoskopik bant ligasyonu ve vazoaktif ilaçlar varis kanamasını durdurmak için kullanılan ana tedavi yöntemleridir. Bazı hastalarda bu tedaviler ile kanama kontrol altına alınamaz. Bu durumda varislere doğrudan bası uygulayarak kanamayı durdurmak amacıyla Sengstaken Blakemore tüpüne (SBT) başvurulur. SBT kanamayı durdurmada oldukca etkili olsa da kullanımı sırasında ölümcül komplikasyonlara neden olabilmektedir. Biz burada SBT kullanımı sonrası özofagus perforasyonu gelişen bir vakayı tartışacağız. (Yoğun Bakım Derg 2013; 4: 28-31)

Anahtar sözcükler: Varis kanaması, balon tamponad, mediastinit

On the other hand, it has some potential complications. While aspiration of secretion is the most common one, acute airway obstruction and esophageal rupture are rare but highly fatal complications (2-4). Herein, we present a case report that had esophageal perforation after a SBT insertion.

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Case Presentation

A 71 year-old woman with a diagnosis of cryptogenic cirrhosis was admitted to the emergency service with hematemesis and hematochezia. Her physical examination revealed tachypnea (22/min), hypotension (80/55 mmHg), ascites, icteric sclera and umbilical hernia. Laboratory examination determined that hemoglobin (hgb) was 6.9 mg/dL; hematocrit (hct) 22.4%; white blood cell count (wbc): 10900/mm³; prothrombin



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time (PT): 22.3 seconds and INR: 2.11. The rest of the physical examination and laboratory tests were normal. Octreotide and proton pump inhibitor (PPI) infusions were started, and after hemodynamic stabilization with saline, packed red blood cells (PRBC) and fresh frozen plasma (FFP), she was moved to the endoscopy unit. The esophagogastroduodenoscopy (EGD) showed grade 3-4 esophageal varices with red spots and oozing, and the presence of fresh blood in the esophagus and stomach. During band ligation, the bleeding increased and only 4 varices could be ligated. The endoscopic procedure had to be stopped early because of severe bleeding causing incapability of band ligation. The patient was intubated to protect the airway. Since she continued to bleed and her condition got worse under treatment of vasopressors and saline infusion, a SBT was inserted urgently but also with difficulty. After confirming the tube placement with auscultation, the gastric balloon was inflated with 300 ml air and the esophageal balloon was inflated to a pressure of about 40 mmHg using a manometer. The tube was fixed to the patient's cheek. Then, the patient was transferred to the medical intensive care unit (ICU). In her past history, she had diabetes mellitus, cryptogenic cirrhosis with grade III esophageal varices and band ligation therapy with no bleeding history.

On arrival in the ICU, she had a Glasgow coma scale of 5, heart rate of 96/minute and blood pressure of 90/50 mmHg, and no active bleeding from the SBT. The rest of her physical examination was the same as the one carried out in the emergency room. She was put on invasive mechanical ventilation and resuscitated with PRBC and FFP. The initial blood tests showed that hgb was 6.7 gr/dL; hct: 20.2%; thrombocytes: 164000 mg/dL; wbc: 20600/mm³; albumin: 2.5 gr/dL; lactate: 2.7 mmol/L; PT: 24.4 seconds; INR: 1.88, activated partial thromboplastin time: 30.1 seconds and total bilirubin: 3.39 mg/dL. The rest of the biochemical tests were normal. Treatment by octreotide and PPI was continued, and ceftriaxon 2 gr q24h and vitamin K were added to the therapy. Child-Pugh score was calculated as grade C and MELD as 19. A chest radiograph taken in the ICU revealed that the gastric balloon of the SBT was located behind the cardiac silhouette (Figure 1). The balloons were deflated and the tube was again passed to the stomach but the patient started bleeding again in a few

minutes. After confirmation of the tube position in the stomach by chest x-ray, the esophageal and gastric balloons were reinflated. Then, she was transferred to the angiography laboratory to have a transjugular intrahepatic portosystemic shunt (TIPS) in the same day. The hepatic venous pressure gradient decreased from 30 mmHg to 20 mmHg and the bleeding stopped after TIPS. A repeated chest x-ray on the second day revealed right chest pneumothorax and a chest tube was introduced into the right hemithorax (Figure 2). Since the diagnoses of the gastric balloon dislocation in the esophagus and right chest pneumothorax had been made, a control EGD was carried out on the third day to see whether the esophagus had been damaged. It showed no sign of bleeding in the esophagus but, ulcers on the ligated varices and a deformed dilated esophagus wall at 30 cm, suggestive of a rupture caused by the SBT. The stomach and proximal part of the duodenum did not have any lesion. Chest computer tomography revealed no sign of evident mediastinitis and esophageal perforation except for right chest pneumothorax, pneumomediastinum and right lung atelectasis (Figure 3). Considering the poor condition of the patient, the decision was made to maintain conservative management with thoracic and general surgery consultations. On the fourth day of admission, fresh blood coming through the nasogastric tube was noticed. The patient was resuscitated with blood products and, fortunately, the bleeding stopped spontaneously. Portal vein Doppler ultrasonography did not show any significant finding including portal vein thrombosis except massive ascitic fluid. On the sixth day, she became hypotensive and had fever. Laboratory studies revealed leucocytosis, high CRP and thrombocytopenia but no fall in hgb level and normal cardiac and ascitic fluid work-ups. Antibiotic treatment was upgraded to piperacillin-tazobactam 4.5 g q6h and ciprofloxacin 400 mg q12h due to suspicion of mediastinitis. Repeated EGD on the same day also revealed a linear esophageal wall rupture between 28 and 34 cm and a metallic stent was placed to close the ruptured area. A chest x-ray confirmed that the stent was in the place (Figure 2). She had fever and hypotension in the following days despite the supportive treatment with vasopressors, fluid and antibiotics, and she died on day 11 of ICU admission. During the stay of the

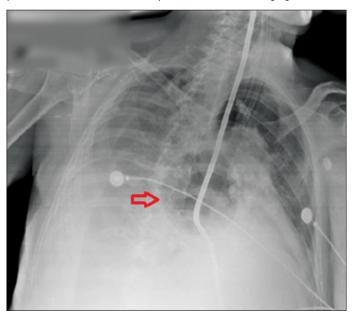


Figure 1. Chest X-ray showing the gastric balloon of the SBT behind the cardiac silhouette (arrow)

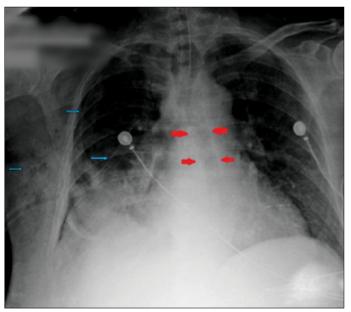


Figure 2. Chest X-ray showing the right chest pneumothorax, subcutaneous air and chest tube (small arrows); metal stent in the esophagus (big arrows)

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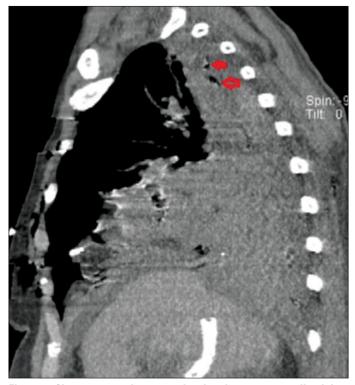


Figure 3. Chest computed tomography showing pneumomediastinium (arrow)

patient in the ICU, 10 units of PRBC and 16 units of FFP were transfused, and all microbiologic cultures were negative.

Discussion

A balloon compression of bleeding varices with SBT is effective in nearly 90 % of cases (2, 3). It is a temporary emergency measure aiming to arrest bleeding, allow volume replacement and prevent further blood loss in order to optimize the patient's condition before definitive managements are planned. It is more effective in patients with less severe hepatic dysfunction and in patients with preceding endoscopic therapy (5). On the other hand, it carries a risk of complications that has been reported to be up to 15% (3). Aspiration of secretions is the most common complication of balloon tamponade (2, 3). Airway obstruction, mucosal pressure necrosis and esophageal rupture are rare but highly fatal complications (2, 4, 6).

A difficult SBT insertion with inflation of the gastric balloon in the esophagus is a major risk factor for esophageal rupture, as seen in our patient. Retching and vomiting lead either to spontaneous esophageal rupture or to dislocation of the gastric balloon to the esophagus which creates pressure sufficient to tear the esophageal wall (6). Repeated sclerotherapy is another risk factor for esophageal rupture by weakening the esophageal wall (7). After insertion of a SBT, auscultation and balloon pressure monitoring alone are not adequate. Therefore, a chest x-ray before and after full gastric balloon inflation is mandatory to confirm correct tube position and detect displacement of the tube back to the esophagus as soon as possible. In case the position of the tube is not able to be labeled by a plain x-ray, the gastric balloon can be filled with radiocontrast material instead of air or water to have an evident radiographic location of the tube (8). The location of the tube can also be confirmed quickly by ultrasonography if available in the service and if the physician has experience in using USG (9). Although it is uncommon, esophageal rupture carries a high incidence of mortality from hemothorax or septic mediastinitis, as demonstrated in our patient. Inflammation and infection can spread rapidly due to anatomical characteristics of the esophagus which lacks serosa. Therefore, mediastinitis and related secondary infections are often encountered after the esophageal perforation.

In our case, the SBT had to be inserted urgently due to severe bleeding of the patient. Since the patient was hemodynamically unstable due to bleeding, the balloons were inflated after confirmation of the tube location with auscultation. In addition, the patient had a difficult tube insertion and a high Child-Pugh classification. All of these are precipitating factors for the esophageal rupture. The diagnosis of the esophageal rupture was confirmed by EGD and conservative management was planned due to the poor condition of the patient. Unfortunately, the patient died because of infection whose source was most probably mediastinitis.

Conclusion

This case demonstrates a fatal complication of SBT insertion even though it is very effective in controlling esophageal variceal bleeding. It shows the importance of radiography in checking the tube position before and after full inflation of the gastric balloon to confirm correct tube position and to detect tube dislocation as soon as possible. If the stomach balloon is inflated in or dislocated to the esophagus, an esophagus perforation should be investigated quickly and a treatment plan should be arranged in case wall rupture is diagnosed.

Informed Consent: Since the ICU period was very painful for the family, a written informed consent was not taken in order not to remind them of their loss.

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