Boerhaave syndrome: A case report

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Spontaneous rupture of the esophagus (Boerhaave syndrome) is a rare condition that has many possible causes, among them violent retching. The definitive treatment for the ruptured esophagus is surgical repair. Potential complications include infection in many forms, pleural effusion, and pneumothorax.

This case study presents an overview of the syndrome, including morbidity, mortality, and treatment. The patient in this case is a 61-year-old man who had 1 episode of violent vomiting resulting in a perforation of the esophagus with communication into the right chest cavity. The patient underwent surgical repair of the rupture with placement of a feeding tube and creation of an esophageal diversion to promote healing of the surgical site.

Key words: **Boerhaave syndrome, esophageal rupture.**

Spontaneous rupture of the esophagus (Boerhaave syndrome) is a rare condition that carries a mortality rate of greater than 30%, even when diagnosed soon after the event and treated aggressively. The definitive treatment is surgical repair. Mortality typically results from subsequent infection in the form of mediastinitis, pneumonitis, pericarditis, or empyema.

This case is a 61-year-old black man with a known history of well-controlled hypertension, well-controlled diabetes, and benign prostatic hypertrophy, who sought care because of right-sided lower chest pain (rated at 10/10) following 1 episode of vomiting. A chest radiograph revealed a right-sided pleural effusion, and, subsequently, a thoracentesis was performed, which resulted in the needle aspiration of gastric contents. An upper gastrointestinal series (with contrast medium) revealed a leak from the lower esophagus with extravasation into the right side of the lower chest.

The patient was taken to the operating room where a right thoracotomy was performed for oversew of the rupture. In addition, an exploratory laparotomy was performed for placement of gastrostomy and jejunostomy tubes, followed by a left-sided neck dissection for development of a "spit" fistula, which is done to divert saliva to the exterior, thereby avoiding exposure of the esophageal surgical repair to saliva for the period needed for healing.

Discussion of the case is presented, followed by discussion of the mechanism of rupture, as well as associated pathophysiologic features.

Case summary

A 61-year-old black man sought care at the emergency department because of acute onset of right-sided lower chest pain following 1 episode of vomiting. Nine hours before the vomiting episode, he had eaten at a Mexican restaurant and subsequently had been nauseated for 6 to 8 hours. He obtained very minimal relief with an effervescent antacid solution. Upon admission to the emergency department, his vital signs were as follows: blood pressure, 133/64 mm Hg; heart rate, 64 beats per minute; respiratory rate, 18 breaths per minute; temperature, 35.6°C (96.1°F); and Sa_{O_2}, 97%. He rated his pain at 10 on a 10-point scale and stated that he had radiation of the pain to his right flank. Routine laboratory data were obtained and revealed the following: white blood cell count, 16 $\times 10^9$/L, and a Pa_{O_2} of 59.5 torr while breathing 100% oxygen via a nonrebreather mask. Table 1 shows the results of arterial blood gas testing. Otherwise, all laboratory data were within normal limits. Results of a 12-lead cardiogram were within normal limits. By emergency department protocol, a 16-gauge intravenous catheter was placed in the right antecubital space and was immediately saline-locked.

The patient's medical history was significant for hypertension controlled with lisinopril; adult onset diabetes mellitus controlled with diet and glyburide; benign prostatic hypertrophy for which transurethral resection of the prostate was performed 12 months before this admission; and right knee surgery in 1969. The patient is a nonsmoker and consumes no alcohol.

A chest radiograph revealed a right-sided lower pleural effusion. The initial diagnosis was aspiration pneumonitis, based on a suspicion that the patient had vomited and aspirated, resulting in a right-sided lower lobe aspiration pneumonitis. Differential diagnoses considered and documented in the patient's history and physical examination record included aortic dissection, esophageal rupture, and Mallory-Weiss tear. Seven hours after admission to the emergency department, the patient was admitted to the intensive care

Table 1. Arterial blood gas results before, during, and after surgical repair

	ED, 9:05 AM	ICU, 2:35 PM	OR, 8:46 PM	ICU, 3:49 AM
Fio_2	1.0	1.0	1.0	0.5
Mode	Face mask	Face mask	Intubated	Intubated
pH	7.31	7.258	7.31	7.34
$Paco_2$ (mm Hg)	42.7	41.8	36.2	35
Pao_2 (mm Hg)	59.5	121.7	131.9	118
Hco_3^- (mEq/L)	21	18.7	17.7	18.6
Base excess	−5.1	−7.9	−7.7	−6.2
Sao_2 (%)	92.3	98.5	99.7	99.5
Gram % oxyhemoglobin	17	18.3	15.8	14.6

ED = emergency department
ICU = intensive care unit
OR = operating room

unit (ICU) in guarded condition with a diagnosis of pneumonia. Normal saline intravenous fluid was initiated at 125 mL/h via the existing 16-gauge catheter.

A chest computed tomography scan after admission to the ICU revealed right-sided pleural effusion, with no free air observed in either the chest or the abdomen. This finding supported the initial diagnosis. A right-sided thoracentesis was performed in the ICU with an immediate return of a "marked amount" of brown fluid with the odor of gastric contents. A pH test of the fluid yielded a result of 5.0. At this point, the diagnosis shifted to Boerhaave syndrome, and an emergency upper gastrointestinal study with contrast medium was undertaken. The results showed a leak of contrast medium from the lower right part of the esophagus with extravasation into the right side of the lower chest cavity. The patient was typed and crossmatched for 4 U of packed red blood cells. A second intravenous line was established (18-gauge catheter) in the left arm to infuse normal saline at 100 mL/h. A radial arterial line and right subclavian central line were inserted while the patient was in the ICU.

The patient was taken to the operating room at approximately 6:30 PM, where, following preoxygenation with 100% oxygen via anesthesia mask and spontaneous ventilation, induction of general anesthesia was accomplished by rapid-sequence induction using midazolam, fentanyl, thiopental sodium, and succinylcholine. Adequate cricoid pressure was applied, and the patient was intubated with a 41F left double-lumen tube via direct laryngoscopy with a MacIntosh 4 blade. Placement of the double-lumen tube was confirmed with fiberoptic bronchoscopy. Maintenance of anesthesia was with 100% oxygen and isoflurane, and muscle relaxation was maintained with *cis*-atracurium infusion at 1 to 2 µg/kg per minute guided by peripheral nerve stimulator.

With the patient in a left-lateral decubitus position, a right-sided thoracotomy was performed with the right lung adequately deflated. The surgeon had difficulty identifying the esophagus due to gastric contents in the field. At the surgeon's request, a 16F Salem sump tube was placed without difficulty, which allowed the surgeon to identify the esophagus while palpating the tube. After the esophagus was identified, aggressive lavage of the chest cavity was performed, after which a 6-mm, round perforation of the lower right side of the esophagus was identified. A T tube was placed in the perforation, attached to bulb suction, and sutured in place, and the Salem sump tube was removed. Incidentally, a right-sided lower lung lobe partial resection was done, with frozen section pathology identifying a benign granuloma. The right lung was reexpanded and checked for leaks. Two chest tubes were placed in the right side, and the chest was closed.

The patient then was positioned supine, and, under direct laryngoscopy, the double-lumen tube was removed and a 7.5-mm orotracheal tube was placed. An exploratory laparotomy then was performed for placement of gastrostomy and jejunostomy tubes, plus the appropriate surgical drains. This portion of the surgery proceeded without incident, and the abdomen was closed.

With the patient remaining in the supine position, his neck extended, and his head rotated to the right, a left-sided neck dissection was done to create a spit fistula via esophagostomy. The surgeon was not able to create the spit fistula due to the inability to mobilize the cervical portion of the esophagus, therefore, a mushroom-tipped catheter was placed into this portion of the esophagus and brought to the surface of the left side of the neck and connected to gravity drainage. The appropriate surgical drains were placed, and the neck then was closed without incident.

The patient was transported to the ICU at 3:00 AM, still intubated, and placed on a volume ventilator. Muscle relaxation was not reversed. Throughout the case, the patient received morphine, 10 mg; fentanyl, 350 µg; midazolam, 3 mg; and thiopental sodium, 400 mg. In the ICU, the patient was sedated for the next 24 hours according to the surgeon's orders.

He was extubated approximately 30 hours after surgery. By postoperative day 8, a right-sided chest empyema had developed that subsequently was drained surgically. He was discharged from the ICU on postoperative day 15.

Discussion

Boerhaave syndrome was first described in 1724.[1-3] The initial description was of a transverse tear in the esophagus; however, the syndrome is more commonly known for a rupture along the longitudinal axis of the esophagus. Anatomically, the rupture is typically on the left side and located on the lower portion of the esophagus, just above the diaphragm.[3] The rupture less commonly occurs in the subdiaphragmatic or upper thoracic area. In the present case, the rupture was on the right side, approximately 2 cm above the diaphragm.

The syndrome is rare, and it is not well reported in the literature. It accounts for approximately 15% of traumatic rupture or perforations of the esophagus.[3,4] Demographically, males suffer this condition more than females, with the greatest percentage in the 40- to 60-year age range.

The esophagus lacks the elasticity of the remainder of the digestive tract. Therefore, the esophagus may rupture with abrupt onset of even relatively low intraluminal pressures, such as that developed with vomiting.[3,5] A similar, albeit not spontaneous condition may be brought about by blunt chest trauma[6] or may be induced iatrogenically in cases of upper gastrointestinal endoscopy or improperly performed cardiopulmonary resuscitation. Most cases of instrumental perforation are small and may occur as a result of dilatation of strictures.[5,7] These nonspontaneous ruptures account for 55% of esophageal ruptures.[3]

Boerhaave syndrome is similar to the more commonly known Mallory-Weiss tear. However, the Mallory-Weiss tear is a shallower tear or laceration (mucosal), whereas the tear in Boerhaave syndrome is an acute rupture (transmural) with communication from the lumen of the esophagus to the pleural cavity. The typical patient with the Mallory-Weiss tear has preexisting esophageal pathology, such as reflux esophagitis or varicosities brought about by alcoholism or portal hypertension. Another distinction is that in the Mallory-Weiss tear, there may be severe bleeding at the site, whereas in Boerhaave syndrome, the blood loss usually is unremarkable.[3]

When the esophagus ruptures, the pain is described as acute chest pain, which may be accompanied by epigastric pain. Because of the pain and its location, patients will have tachycardia and tachypnea when they are admitted. There may be blood loss; however, it usually is unremarkable.[3] Breath sounds are diminished on the side of the perforation as pleural effusion develops. There may be accompanying subcutaneous emphysema. The Mackler triad (vomiting, chest pain, and subcutaneous emphysema) is diagnostic; however, the triad is quite uncommon.[3]

In 90% of cases, a chest radiograph reveals pleural effusion; however, this is not necessarily diagnostic.[3] Other pathology present on a chest radiograph may be mediastinal emphysema, pneumothorax, or pneumomediastinum. In some cases, the chest radiograph may be normal, such as when there is little or no extravasation of gastric contents into the pleural cavity. A chest tube may be needed to evacuate gastric contents from the pleural cavity, as well as to reexpand the lung.

Results of upper gastrointestinal studies such as contrast medium swallows usually show extravasation but may have false-negative results in up to 20% of patients.[3,4]

Before 1947, the syndrome was almost universally fatal. In that year, Barrett described the first successful surgical intervention.[8] Since then, surgery to repair the rupture has become the definitive treatment, although instrumental pe rforations can be managed nonsurgically.[4,7] Even if treated promptly, the mortality approaches 50%, usually related to sepsis, mediastinitis, pericarditis, pneumonitis, or empyema.[2-4] There also exists the risk for recurrent spontaneous rupture, although this complication is rare.[9] Unfortunately, because of the rarity of the condition, the diagnosis frequently is missed. If the rupture is untreated, the mortality is near 90%.

Thoracotomy with aggressive lavage and repair of the rupture is the preferred treatment. In this case, the gastrostomy and jejunostomy tubes were placed for long-term nutritional support, and the mushroom catheter was exteriorized from the cervical esophagus to divert saliva. The use of a nasogastric tube postoperatively is controversial, since its presence is likely to increase gastroesophageal reflux, which is considered detrimental to healing.[7] In this case, the nasogastric tube was placed only for "landmark" purposes intraoperatively and then removed. Table 2 shows anesthetic considerations for patients with Boerhaave syndrome.

Conclusion

Boerhaave syndrome is a rare condition that presents the anesthetist with a challenging, emergency situation. Even in the light of rapid diagnosis and aggressive intervention, the mortality rate approaches numbers rarely seen in other surgical emergencies.

Table 2. Anesthetic concerns for the patient with Boerhaave syndrome

Preoperative
Large-bore intravenous access
Fluid replacement of preoperative losses (ie, vomiting)
Antibiotics
Age-specific and comorbidity-warranted tests
Intraoperative
Rapid-sequence induction and intubation with cricoid pressure
Double-lumen tube and one-lung ventilation on 100% oxygen
Fiberoptic confirmation of double-lumen tube placement
Avoidance of esophageal instrumentation
Minimal blood loss
Warming measures (air warmer, intravenous fluid warmer, heated humidifier)
Positioning concerns
Postoperative
Possible need for postoperative mechanical ventilation
Single-lumen endotracheal tube for mechanical ventilation
Assess for recurrent laryngeal nerve injury caused by surgical exposure of cervical esophagus
Pain relief and control
Aggressive pulmonary toilet

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