Boerhaave's Syndrome:

A Review of Six Cases of Spontaneous Rupture of the Esophagus Secondary to Vomiting

WILLIAM O. BOBO, M.D., WILLIAM A. BILLUPS, M.D.,
JAMES D. HARDY, M.D., F.A.C.S.

From the Department of Surgery, University of Mississippi Medical Center and University Hospital, Jackson, Mississippi 39216

THE TERM "spontaneous rupture" of the esophagus is something of a misnomer, but it is generally used to designate lesions resulting from intrinsic forces acting upon the esophagus as opposed to trauma. Excluded also are perforations associated with carcinoma, lye strictures, or other esophageal disorders. The first account of spontaneous perforation appeared in 1724 when Boerhaave [4] described the classic case of Baron John van Wassenaer, Grand Admiral of Holland. The Baron, who suffered from indigestion, sought to "cleanse the stomach gently" by vomiting, and in the attempt sustained a ruptured esophagus. He died 18½ hours later, and at autopsy Boerhaave found a tear in the distal esophagus with food material in both pleural cavities. Since that description more than 200 instances of spontaneous perforation have been reported. Discussion of the pathophysiology and natural history were published by Anderson [1] in 1952 and Derbes and Mitchell [7] in 1956. Successful thoracotomy in management was first performed by Barrett [2] in London in 1946. He closed the esophageal tear with one purse string suture of catgut. Although a small leak with mediastinal abscess developed postoperatively, the patient recovered. At that time there were some 50 cases reported and apparently only three patients survived after surgical drainage.

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Prior to surgical treatment the mortality rate approached 100%, as reported in the review by Derbes and Mitchell [7] in 1956. Of those treated without operation, 35 per cent were dead in 24 hours, and all were dead within one week. Since the advent of operative treatment 60 to 70 per cent of patients survive. The high morbidity that accompanies surgical intervention is illustrated in a report by Briggs *et al.*[5] They noted that all of a series of eight patients who had thoracotomies for ruptured esophagus developed empyema, and in four suture lines broke down with development of esophagopleural fistulas. Derbes and Mitchell reported esophagopleurocutaneous fistulas in 19 of 41 patients who survived 5 days after thoracotomy.

Hardin *et al.*[9] in 1967 reviewed all esophageal perforations at the Hospital of the University of Mississippi of which only two were spontaneous ruptures. One was discovered at autopsy of a patient who had prolonged bleeding from a duodenal ulcer and esophageal rupture was a terminal event. The other patient, a chronic alcoholic, was seen 2 days after a rupture while vomiting during a drinking bout. The diagnosis was made by barium swallow x-ray, but because his condition was stable, thoracotomy was not performed. He was given antibiotic therapy and was fed through a Miller-Abbott tube passed into the jeju-

num. X-rays showed the defect 2 weeks after perforation, but the patient recovered uneventfully. Since that time four cases of spontaneous esophageal rupture have been seen. One patient suffered a cardiac arrest in the emergency room 5 minutes after arriving in profound shock. A two-centimeter perforation in the distal esophagus was demonstrated at autopsy. The other three were successfully treated by thoracotomies, and findings in pre-, intra- and postoperative courses are discussed under those headings.

Preoperative Course

Our patients were men, 50 to 65 years of age. Data accumulated support Anderson's concept that the esophageal wall may have been weakened by pre-existing peptic esophagitis, alcoholism, or poor nutrition. For example, in Berne's series,[3] two of five patients had co-existing peptic ulcer disease and three were alcoholics. Of our six patients, one was alcoholic, one had bleeding duodenal ulcer disease, another had chronic indigestion and one was a moderately heavy drinker. The fifth was in good health, and no history was obtainable on the sixth. Effort rupture may occur with sudden increases in intra-abdominal pressure, including seizures, coughing, parturition, defecation, and heavy lifting. Rupture is usually the result of vomiting, however, as in all six of our patients, characteristically following ingestion of a large amount of liquid or food. One patient drank a large volume of ice water, another had a glass of cold milk after consuming a large quantity of beer, and a third vomited after drinking a cup of coffee one hour after lunch. All had sudden excruciating lower left chest and epigastric pain after vomiting.

Intense mediastinitis and pleuritis (chemical, enzymatic, and subsequently bacterial) from contamination with esophageal and gastric contents may lead to vascular collapse, as in a patient who died in the

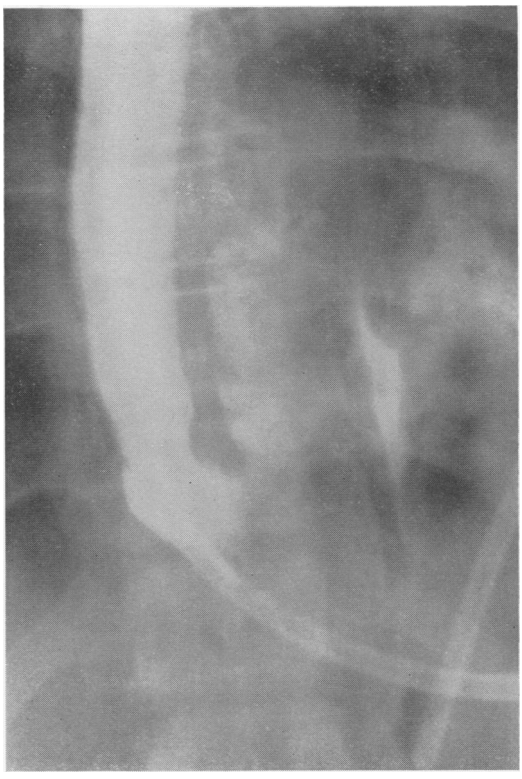

FIG. 1. Barium esophagogram demonstrating leakage of dye into left hemithorax.

emergency room. None of the four who survived were hypotensive on admission, though the three who were operated upon were in respiratory distress and had tachycardias of 120–130. In this regard the interval between onset of symptoms and the initiation of treatment is critical. Three operative patients arrived 2, 5 and 9 hours after onset of pain, and the operations were begun 9, 9½ and 16 hours after onset.

Most reports indicate that Anderson's triad of subcutaneous emphysema, rapid respirations, and abdominal rigidity is not universal. Dryness and grunting respirations occurred in all our patients. Two of three operated upon had decreased breath sounds on the left, and chest x-rays showed hydropneumothorax (30 and 70%) in both. The third patient, whose chest was clear was the only one who had air in the soft tissues of mediastinum and neck. The

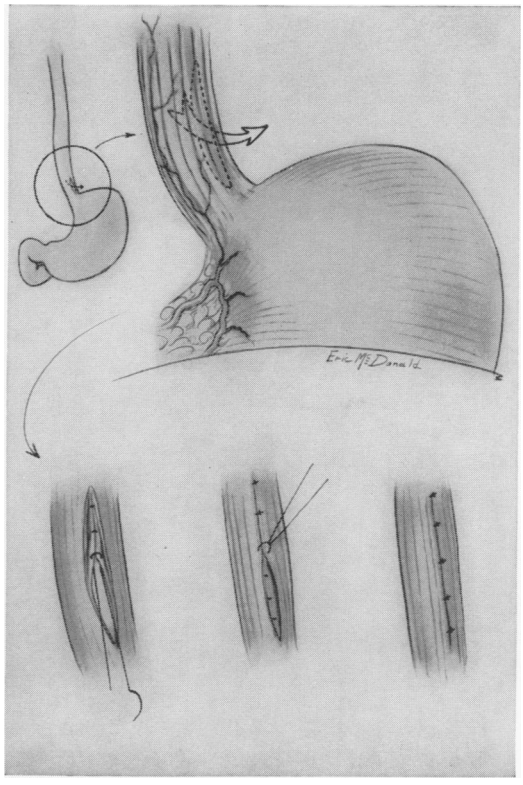

FIG. 2. Illustration of the usual site of spontaneous rupture of the esophagus in the left posterolateral wall just above the diaphragm. Below is shown the method of repair.

diagnosis may hinge on recognizing a very small amount of mediastinal air. Epigastric tenderness and rigidity with decreased bowel sounds was a feature in all three patients. The diagnoses were confirmed by barium swallow x-rays.

Intraoperative Course

Left lateral thoracotomies through the bed of the eighth rib were performed in all three cases. Spontaneous rupture, in contrast to other perforations, is usually explosive and accompanied by rupture of the mediastinal pleura as in two of our patients. Leakage was confined to the mediastinum in the third though he had 600–700 cc. serosanguineous fluid in the left hemithorax at operation. The esophageal lacerations were longitudinal, 2.5 to 3 cm. in length, and located just above the hiatus of the diaphragm. Two were left anterolateral and one left mediolateral, in contrast to the usual posterolateral reported. Only one laceration had necrotic edges requiring debridement. The method of closure in all three was with interrupted 3-0 silk sutures placed 5–7 mm. apart with knots inside the lumen to close the mucosa, and muscularis with interrupted 3-0 silk sutures. The mediastinal pleura was left open for drainage, and drainage tubes were placed in the area of the mediastinal rent.

Postoperative Course

Atelectasis, retained secretions and pneumonitis developed in all patients. Sputum cultures grew *E. coli* and Proteus from one, and Pseudomonas and Klebsiella aerobacteria from another (Table 1). The latter patient developed empyema which was treated with Varidase for 1 week then converted to open drainage on the 17th postoperative day. Varidase in the presence of a fresh suture line caused concern. However, Varidase acts principally on fibrin and purulent material, and it caused no ill effect on the esophageal repair. The empyema resolved satisfactorily within 6 weeks. Another patient had a mild empyema which grew out *E. coli* freundii, but responded to antibiotic therapy. The chest tube was removed by the 12th postoperative day.

One patient sustained a period of hypotension during operation which responded to blood transfusion. Postoperatively, however, he developed acute hepatic failure which progressed to coma. Serum bilirubin rose to 25 mg./100 ml. on the 6th postoperative day, but he recovered. During this time he was also treated for cardiac arrhythmias and congestive heart failure.

The two most recent patients were given intravenous hyper-alimentation. All three were discharged in satisfactory condition and were well thereafter.

TABLE 1. *Complications of Operated Cases*

Patient	Complication	Organism	Source	Antibiotics
E. S.	1. Pneumonitis (bilat.) 2. Slight left empyema 3. Hepatic failure 4. Congestive heart failure	*E. coli freundii*	Chest tube (sputum negative)	Penicillin Streptomycin Colymycin
G. W.	1. Pneumonia 2. Wound infection 3. Empyema	Pseudomonas Klebsiella a.	Chest tube Sputum Sputum	Penicillin Kanamycin Colymycin Polymixin Ampicillin-IV + PO
J. B. L.	1. Pneumonia (RU + LL)	*E. coli* Proteus	Sputum	Keflin IV Keflin (in thorax at operation) Ampicillin PO

Discussion

Occasionally a patient survives spontaneous esophageal rupture without operation. It is impossible to determine early in the disease which patient will survive, even when the lesion is confined to the mediastinum. In view of the mortality without operation, early operation is indicated. Initial therapy is directed toward preparing the patient for operation.

Replacement of intravascular loss with plasma, electrolyte solutions, or blood is begun initially. In some this loss may cause shock. Hydropneumothorax is relieved with intercostal chest tubes. Hypoxia may be a threat at this point. Tracheostomy was not necessary in our cases. A nongastric tube is inserted preoperatively to prevent further contamination of the thorax with gastric contents. Large doses of antibiotic drugs are given preoperatively. Corticosteroids in large doses (250–500 mg. Hydrocortisone or its equivalent every 4–6 hours) intravenously for 48 to 72 hours may reduce the inflammation in the mediastinum and pleura, though this is uncertain.

Berne [3] advocated the trans-abdominal approach for supracardial ruptures, supplemented by intercostal drainage, as an advantage in buttressing the esophageal repair with gastric fundus and treating coexisting peptic ulcer disease by vagotomy and gastric drainage. All patients recovered, although two repairs leaked postoperatively. Unless there is a specific indication for laparotomy, such as free air or extravasation of radiopaque material beneath the diaphragm, we favor the thoracic approach. It provides direct exposure of the structures and access to the thoracic cavity for irrigation and placement of drainage tubes. Necrotic wound edges should be debrided. We did not find it necessary to buttress the repair or to use onlay gastric patches as proposed by Hatafuku and Thal.[10, 11] Such methods may be valuable when the esophageal lumen is compromised or when leakage or stricture are anticipated. Necrosis of esophageal tissue may require wide debridement, or friable tissue may prevent adequate repair. The interval between rupture and repair is one of the most important factors.

Sepsis has been a major cause of death. Repeated cultures of sputum and chest tube drainage are taken during the postoperative period. Bronchoscopy may be beneficial in atelectasis and pneumonitis.

Summary and Conclusions

Six cases of spontaneous rupture of the esophagus secondary to vomiting have been reviewed. Emphasis has been placed on three successfully treated by thoracotomies. Two patients died before operation could be attempted, and one patient survived without operation. The diagnosis was made on plain chest x-ray and confirmed by barium esophagography. Repair was effected with two layers of interrupted silk, inverting the mucosal layer, and no leaks developed. Wide drainage and massive antibiotic therapy were employed. Intravenous hyperalimentation may have been beneficial during the immediate postoperative period. Oral alimentation was begun after removal of the nasogastric tube on the seventh to ninth postoperative day. Pneumonitis was the prevalent complication. Empyema was a complication in one case, and Varidase was beneficial without harm to the esophageal repair.

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