Spontaneous Rupture of the Esophagus:
Boerhaave's Syndrome

HOOSHANG BOLOOKI,* M.D., F.R.C.S.(C), IAN ANDERSON,** M.D.,
CARLOS GARCIA-RIVERA,** M.D., JAMES R. JUDE,*** M.D., F.A.C.S.

From the Division of Thoracic and Cardiovascular Surgery, University of Miami School of Medicine, Miami, Florida 33136

IN 1724 Boerhaave reported a case of postemetic rupture of the esophagus following overindulgence in food and alcohol. The diagnosis was made at autopsy.[6] This clinical entity is also known as effort rupture or postemetic rupture of the esophagus and is characterized by: (a) acute upper abdominal and thoracic pain which is commonly preceded by (b) episodes of vomiting and (c) mediastinal and cervical subcutaneous emphysema. The pathologic lesion is due to direct contamination of the mediastinum and the thoracic cavity with the acid peptic juice and gastric contents.

Since the first recorded successful repair by Barrett in 1947[3], limited individual experiences have been reported and in spite of emphasis on prompt diagnosis and immediate operation an overall mortality rate of 35% prevails.[8] In untreated patients the mortatlity is 50% within 24 hours and 90% within 48 hours.[1, 8]

The following report outlines our method of management based on experience with six patients encountered in the past 18 months. Clinical courses are summarized in Table 1. Important diagnostic points are emphasized and a modification of the technic of closure of the esophageal tear is presented in detail. Results indicate that with careful surgical management mortality and morbidity can be lowered to acceptable levels.

Submitted for publication August 27, 1970.

* Assistant Professor of Surgery and Physiology.

** Resident, Thoracic and Cardiovascular Surgery.

*** Professor and Chief of Division.

Diagnosis

Patients invariably complain of upper abdominal and lower thoracic pain associated with abdominal guarding, rigidity and shortness of breath. Mediastinal emphysema is always present and as time passes cervical subcutaneous emphysema develops. Suspicion is raised on the basis of clinical history and findings on physical examination. The diagnosis is usually suggested on x-ray films taken in postero-anterior and lateral decubitus positions (Fig. 1A and B).

In our patients the diagnosis was always confirmed by Gastrografin esophagograms taken in the lateral decubitus position (Fig. 2). This was done during preparation and waiting for operation. Upright abdominal films were also obtained to rule out extension of the tear into the fundus of the stomach and into the lesser omental sac.

Management

A naso-gastric tube was passed on admission. There was no concern that a straight Levine tube might pass through the tear into the thoracic cavity, as the area of rupture is longitudinal, usually on the left side and posterior. In addition, the

TABLE 1. *Spontaneous Rupture of Esophagus (Boerhaave's Syndrome)—Jackson Memorial Hospital 1/1/69-7/1/70*

Case	Age,	Sex	Interval Rupture to Surgery	Clinical Picture on Admission	Diagnosis Made By	Surgical Treatment	Complications	Discharge
1	61	M	24 hours	hypotension stuporous	physical examination, esophagogram	direct closure and chest tubes	fluid collections in chest	18 day
2	28	M	12 hours	pain, abdominal catastrophe	esophagogram	initial chest tube and direct closure	fluid collections in chest	21 day
3	34	M	30 hours	shortness of breath, abdominal pain	esophagogram	direct closure chest drainage	none	10 day
4	72	M	3 days	shortness of breath, cervical subcutaneous emphysema	physical examination, chest x-ray, esophagogram	direct closure and chest drainage	azotemia pneumonia	20 day
5	37	M	16 hours	shortness of breath, abdominal pain	esophagogram	direct closure and chest drainage	none	10 day
6	59	M	14 days	fever, septicemia collection in the chest. No trouble with swallowing!	physical examination, found mediastinal empyema. Methylene blue test.	mediastinal drainage twice, feeding gastrostomy	empyema mediastinitis	60 day

edges of the longitudinal tear are usually opposed unless there is an increase in intraluminal pressure (i.e. during vomiting). The incident is usually preceded by heavy consumption of food or alcohol. Gastric lavage, therefore, may be necessary although usually gastric contents have emptied into the chest cavity. Occasionally the stomach has been lavaged in the operating room after closure of the rupture.

Intravenous fluid replacement has been necessary to correct hypovolemia and hypotension. The amount of fluid was guided by urinary output, central venous pressure and especially pulse rate and hematocrit. The volume of fluid required has been of the same magnitude required by patients with acute pancreatitis or peritonitis.[7] Large volumes of plasma, Dextran or albumin were given, supplemented by crystalloid solutions. Closed drainage of the chest cavity prior to operation is not essential unless the parietal mediastinal pleura is torn and there is hydro-pneumothorax. Thus, if operation was planned within an hour tube drainage of the chest was delayed.

The time interval between perforation and operation has a direct effect on outcome as does the degree of thoracic contamination.

Operative closure was performed as soon as the diagnosis was made. In Cases 2 and 6 the diagnosis and operation were delayed for 12 hours and 14 days, respectively. In these the progressively deleterious course of the disease was obvious. In Case 2 contrast roentgenograms taken at another hospital 7 hours after onset of symptoms showed the mediastinal emphysema and a small esophageal leakage (Fig. 3A). Contrast x-rays taken 12 hours later at our hospital showed extension of the rupture through the mediastinal parietal pleura contaminating the left thoracic cavity (Fig. 3B and C). Similarly in Case 6 misdiagnosis of the rupture and delay caused the development of a mediastinal

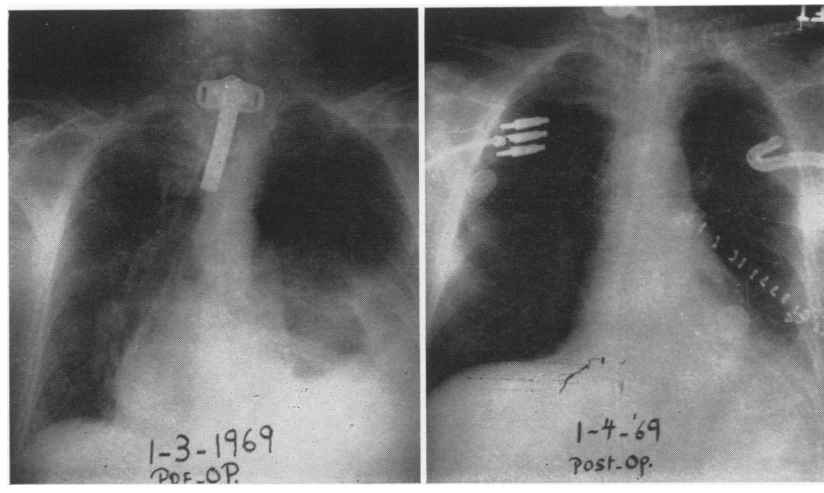

FIG. 1A. Preoperative chest x-rays in Case 1 show the presence of air and fluid in the left chest and the mediastinal emphysema with its shift to the right. B. Chest x-ray one day after operation. Note position of the chest tubes. The upper chest tube was removed on the 2nd day. A fluid collection on the 10th day after operation was noted and subsided with thoracentesis.

abscess and empyema in spite of inadequate tube drainage. The usual sequence is rupture of the mucosal and muscular layers of the esophagus with leakage and contamination of the mediastinum followed by rupture of the mediastinal pleura, chemical mediastinitis and pleuritis. In time these processes progress to a large empyema.

Surgical Technic

In acute cases the site of rupture was exposed through a standard left lateral or anterior thoracotomy. Necrotic tissue (mediastinal pleura) surrounding the tear was debrided. Early in our experience it was realized that the length of the tear in the muscular layer was shorter than that in the mucosal layer. For this reason the opening in the muscular coat was extended one to two centimeters superiorly and inferiorly to visualize the ends of the mucosal rupture (Fig. 4A–E). At times the muscularis tear was lengthened to the level of the diaphragm. Neglect of this point results in postoperative leakage after repair in some patients.[5] The esophagus was repaired with fine silk (0000) in two layers. The edges of the tear were not held with forceps and sutures were passed without tension on esophageal tissue. Sutures were tied gently and not tightly. Thereafter, the chest cavity was washed with 5 to 6 liters of saline solution containing 1–2 Gm. of Kanamycin. Large bore Tygon tubes (20 mm. diameter) were used for drainage to evacuate air and fluid. The quantity of drainage occasionally exceeded 1,000 ml. /day. This fluid loss was replaced with colloid solutions. Large draining tubes permitted good expansion of the lung and

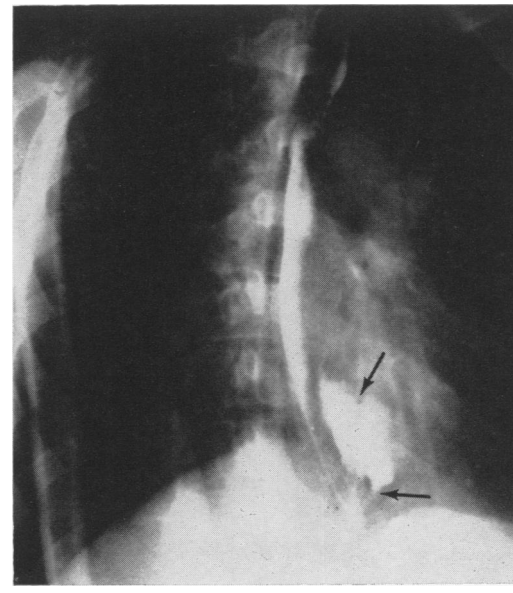

FIG. 2. Case 3, left lateral decubitus film taken during Gastrografin swallow shows evidence of leakage of contrast from the lower esophagus.

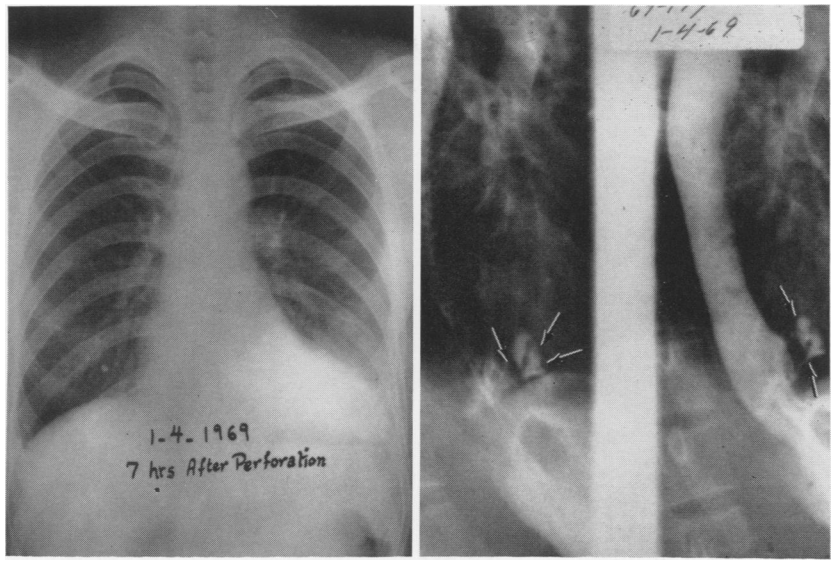

FIG. 3A. Plain x-ray view of the chest from Case 2 seven hours after spontaneous rupture of the esophagus. Note, slight blunting of the left costodiaphragmatic angle. B. Gastrografin esophagogram taken at the left lateral decubitus position shows leakage of contrast material at the left lower esophageal area (arrows).

obliterated dead space. Abdominal incision has not been used.[5]

In instances when the lesion was recognized days after the rupture (as in our Cases 4 and 6), operative procedures were carried out as soon as the diagnosis was established. Delayed closure (Case 4) or drainage of a mediastinal abscess without repair of perforation were performed (Case 6). In this patient, due to inflammation and abscess, the edges of the tear were ragged and edematous and impossible to suture. Drainage with feeding gastrostomy were performed with eventual spontaneous healing. It was not necessary to use T-tubes as described by others.[11]

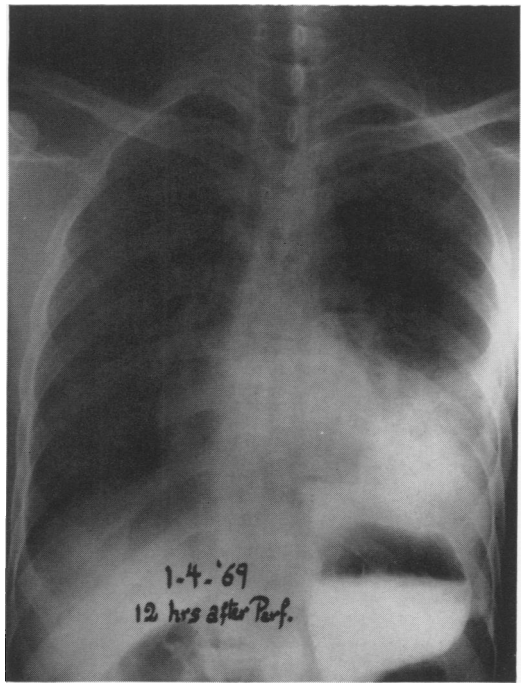

FIG. 3C. Plain P-A x-ray taken 12 hours after esophageal perforation shows a large collection of fluid in the left chest with mediastinal emphysema.

Postoperative Management

Patients were given massive doses of intravenous Penicillin and Streptomycin for 5 days, starting prior to operation. Two large bore chest tubes were used in every case; an upper in the 2nd space midclavicular line and a lower near the repair site through the 7th or 8th interspace at the mid axillary line. These remained in place for at least 4 days.

For 2–3 days after operation tachycardia, tachypnea and mild hemoconcentration were treated with fluid administration of up to 6 liters per day including at least 1500 ml. of plasma or albumin. By the fourth day Gastrografin x-rays showed closure site intact in all acute cases. The nasogastric tube was then removed. Oral fluids were given on the fifth postoperative day except in Case 6 in which gastrostomy

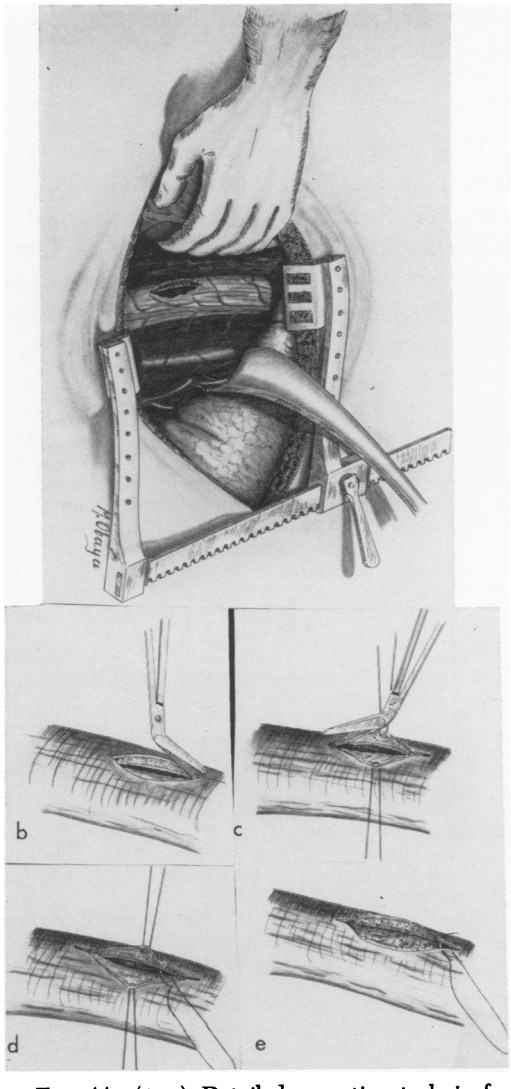

FIG. 4A. (top) Detailed operative technic for closure of spontaneous rupture of the esophagus as seen through a standard left anterior thoracotomy. b and c: Due to the larger mucosal tear, the muscular tear is extended upwards and down to the phreno-esophageal ligament in order to visualize the entire length of the esophageal tear. d and e: The esophagus is then closed in two layers using 0000 cardiovascular silk sutures.

feedings were given on the fifth day and intravenous antibiotics were continued for 10 days.

Chest tubes were usually removed on the fifth and seventh days except in Cases 1 and 2 which are discussed below. Surgical complications occurred in two patients. Both had fluid collections in the chest on the eighth and tenth postoperative days (Case 1 and 2). In Case 1, this was treated with thoracentesis, in Case 2 by enzyme debridement (Varidase). In each case chest x-rays were clear within 2 weeks after the last aspirations. These collections were probably the result of early removal of the chest tubes.

Postoperative respiratory care was that used in the surgical intensive care unit.[4] Physiotherapy, nasotracheal suction and positive pressure breathing in the first 3 days were employed. In all patients cineradiographic studies of the esophagus were performed during a water swallow test prior to discharge. An upper gastrointestinal x-ray was taken to rule out coincidental peptic lesions.[5]

Discussion

This syndrome is thought to be similar to or identical with, the Mallory-Weiss Syndrome.[9, 12] In 1932, Mallory and Weiss reported 21 patients with postemetic tearing of the gastroesophageal mucosal junction causing upper gastrointestinal bleeding.[14] In this condition the mucosal tear is superficial and does not extend through the muscular layer. The tears may be multiple and characteristically cause bleeding. Differential diagnosis from Boerhaave's Syndrome is important since the latter requires immediate operation. In the Mallory-Weiss Syndrome however, the need for operative intervention either for diagnosis or for control of bleeding is not great and conservative management suffices.

The pathogenesis of both Boerhaave's and Mallory-Weiss Syndromes is forceful vomiting with a sudden rise in esophageal intraluminal pressure. During vomiting, the intragastric pressure rise may approximate 120 mm. Hg. Experimentally, in a fresh cadaver, with the esophagus cross clamped at mid portion, a pressure of 150 mm. Hg will tear the gastroesophageal junction, usually on the left side.[13] It is conceivable that with lower pressures only the mucosal layer tears to produce the Mallory-Weiss Syndrome. The tear is almost always longi-

tudinal, along the line of least resistance at the gastroesophageal junction. In both instances pre-disposing factors have been listed such as hiatal hernia, peptic esophagitis and gastric reflux.[2, 10] The reported incidence of Boerhaave's Syndrome has a male/female ratio of 5 to 1. It usually occurs in the fourth to sixth decades of life. A history of vomiting blood is not unusual.[11]

The differential diagnosis is based on physical examination, evidence of contamination of the thoracic cavity, the severity of pain and presence of mediastinal and subcutaneous emphysema. An upright chest x-ray and an esophagogram taken in the lateral decubitus and upright positions confirm the diagnosis of Boerhaave's Syndrome.

Morbidity and mortality should be nil, if recognized early in the course.

In our experience complications are dependent on:

(a) Length of time between rupture and operative intervention.
(b) Extent of contamination of the thoracic cavity and the nature of soilage.
(c) Adequacy of mediastinal and chest drainage.
(d) Adequacy of plasma, fluid and electrolyte replacement. Persistence of tachycardia and elevated temperature are guides to the amount of fluids to be administered.

Summary

Experience with six cases of postemetic rupture of the esophagus (Boehaave's Syndrome) is presented. Differentiation from the Mallory-Weiss Syndrome, which rarely requires operative intervention is essential. Careful postoperative fluid and volume replacement, meticulous repair of the mucosal and muscular layers of the esophagus and adequate drainage of the thoracic cavity and mediastinum are the main safeguards against undue morbidity and mortality in early and in delayed cases. The plan of management is discussed. All our patients recovered.

Addendum

The clinical course of Case 3 was presented at the 55th Annual Clinical Congress of American College of Surgeons (October 1969, San Francisco) in the form of a movie which can be obtained from the Film Library of the American College of Surgeons.

References

1. Abbott, O. A., Mansour, K. A., Logan, W. D., Jr., Hatcher, C. R., Jr. and Symbas, P. N.: A Traumatic so-Called "Spontaneous" Rupture of the Esophagus: A Review of Forty-Seven Personal Cases with Comments on a New Method of Surgical Therapy. J. Thorac. Cardiovasc. Surg., **59**:67, 1970.
2. Baglio, C. M. and Fattal, G. M.: Spontaneous Rupture of the Stomach in the Adult. Amer. J. Dig. Dis., 7:75, 1962.
3. Barrett, N. R.: Report of a Case of Spontaneous Perforation of the Esophagus Successfully Treated by Operation. Brit. J. Surg., **35**:216, 1947.
4. Bendixen, H. H., Egbert, L. D., Hedley-Whyte, J., Laver, M. D. and Pontoppidan, H.: Respiratory Care. St. Louis, C. V. Mosby Co., 1965, pp. 93–103.
5. Berne, C. J., Shader, A. E. and Doty, D. B.: Treatment of Effort Rupture of the Esophagus by Epigastric Celiotomy. Surg. Gynec. Obstet., **129**:277, 1969.
6. Boerhaave, H.: Atrocis, nec descriptiprius, morbi historia. Secundum medicae artis leges conscripta. Lugduni Batavorum, Boutesteniana, 1724.
7. Bolooki, H., Jaffe, B. and Gliedman, M. L.: Pancreatic Abscesses and Lesser Omental Sac Collections. Surg. Gynec. Obstet., **126**: 1301, 1968.
8. Bruno, M. S., Grier, W. R. and Ober, W. B.: Spontaneous Laceration and Rupture of Esophagus and Stomach. Mallory-Weiss Syndrome, and Their Varients. Arch. Intern. Med., **112**:574, 1963.
9. Byrne, J. J.: The Mallory-Weiss Syndrome. New Eng. J. Med., **272**:398, February 25, 1965.
10. Conte, B. A.: Esophageal Rupture in Absence of Vomiting. J. Thorac. Cardiovasc. Surg., **51**:137, 1966.
11. Hinchey, E. J. and Hreno, A.: Post-Emetic Gastroesophageal Laceration with Hemorrhage. Surg. Gynec. Obstet., **126**:324, 1968.
12. Holmes, K. D.: Mallory-Weiss Syndrome: Review of Twenty Cases and Literature Review. Ann. Surg., **164**:810, 1966.
13. Mackler, S. A.: Spontaneous Rupture of the Esophagus an Experimental and Clinical Study. Surg. Gynec. Obstet., **95**:345, 1952.
14. Mallory, G. K. and Weiss, S.: Hemorrhages from Lacerations of the Cardiac Orifice of the Stomach due to Vomiting. Amer. J. Med. Sci., **178**:506, 1929.