Mallory-Weiss Syndrome and Emetogenic (Spontaneous) Rupture of the Esophagus *

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TWO CASES of emetogenic rupture of the esophagus have been seen on the First (Columbia) Surgical Division at Bellevue Hospital since 1944. One patient was admitted for bleeding peptic ulcer. The other had what we believe was Mallory-Weiss syndrome and rupture of the esophagus which followed continued hematemesis in the hospital.

In 1724, Boerhaave [3] described the case of the Grand Admiral of Holland, Baron van Wassenaer. The Baron, following his usual overindulgence at the dinnig table, and subsequent to self induced emesis, experienced severe chest pain, dyspnea, and cyanosis and died 18 hours later. Autopsy disclosed bilateral empyema, mediastinal emphysema and a fresh rupture in the distal third of the esophagus. In 1877, Fitz [10] reviewed the literature on this subject, but it was not until 1947 that Barrett [1] and Olsen and Clagett [17] successfully treated the disease surgically.

In 1929 and 1932, Mallory and Weiss [15] reported 21 cases of gastroesophageal bleeding with hematemesis. Six died, five from exsanguination and one from rupture of the esophagus. The five who bled had two to four linear lacerations of the mucosa and submucosa of the cardiac portion of the stomach with extensions into the terminal esophagus in most of the cases.

The pathologic nature of spontaneous rupture of the esophagus and possible etiology have been discussed at length.[1, 5, 7, 8, 9, 11, 19] In brief, the rupture is most often just above the diaphragm on the left posterolateral wall, is longitudinal in direction and is from 1 to 8 cm. in length. Experiments indicate [13] that 3 to 5 pounds pressure per square inch in the isolated esophagus will cause a longitudinal rent through all layers at about the same area in which similar rents are seen clinically. Bodi, Sanger and Forsythe [2] showed that on the basis of physical principles less tension is required to cause rupture in a longitudinal than in a transverse direction. In the case reported by Boerhaave the rupture was circular, and rare transverse ruptures have been reported by others.

Small and Ellis [21] in 1958 for the first time related spontaneous rupture of the esophagus to the Mallory-Weiss syndrome, stating that vomiting caused both conditions.

There has been considerable confusion with respect to the mechanism of the Mallory-Weiss syndrome and spontaneous rupture of the esophagus (Table 1). Both injuries appear to be the result of abnormally elevated intraluminal cardioesophageal pressures during vomiting. Lack of neuromuscular coordination during vomiting leads to unphysiologic pressures. In the Mallory-Weiss syndrome the injury is milder and is manifested by hemorrhage from lacterations of the gastroesophageal mucosa. On the other hand, rupture is complete laceration of the distal esophagus through all layers and represents more severe explosive injury. The force of this

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TABLE 1. *Comparison of Mallory-Weiss Syndrome with Emetogenic (Spontaneous) Rupture of Esophagus*

Characteristic	Mallory-Weiss Syndrome	Emetogenic (Spontaneous) Rutpure of Esophagus
History	Forceful vomiting or retching (often recurrent)	Same
Associated conditions	Alcoholic introxication, central nervous system lesions or exhaustion	Same
Etiologic mechanism	Excessive intraluminal pressures during uncoordinated vomiting	Same
Symptoms & signs	Painless massive hematemesis & possible shock	Back, chest, and/or epigastric pain. Dyspnea. Cyanosis. Possible hematemesis. Possible shock. Pleural effusion. Subcutaneous emphysema. Mediastinal, respiratory or cardiac crunch (Hamman's sign)
Pathology	Longitudinal mucosal & submucosal lacerations	Longitudinal laceration through all layers (rarely transverse)
Location	Esophagogastric junction	Distal esophagus above gastroesophageal junction (rarely below the diaphragm)
Clinical course	Death from hemorrhage if not controlled	Shock. Possible hemorrhage. Mediastinitis. Pleural effusion. Pneumothorax. Empyema. Rapid deterioration. Fatal if not treated
Overall mortality	0–25%	30–70% (in various series)

explosion and gastric contents at the time determine the gravity of the disease and may explain some discrepancies in reported mortality rates. Since rupture of the esophagus is not truly spontaneous, we propose the term *emetogenic rupture* as a substitute. This term relates the disease to its immediate cause and also may clarify semantic confusion.

The marked toxicity and circulatory collapse which often accompany esophageal rupture are related to mediastinal compression, severe chemical and bacterial mediastinitis and pleuritis. Later in the disease, endotoxin shock may play a role in the inevitably stormy course. Ware *et al.*,[22] as well as Bruno and colleagues,[4] note the high mortality associated with emetogenic rupture of the esophagus. In the collected series of Ware, 68 of 86 patients (79%) died. The following two cases represent severe emetogenic rupture of the esophagus, or, as Bruno suggested, Boerhaave's Syndrome.

Case Reports

Case 1. A 46-year-old white man was admitted with the chief complaint of melena of 18 hours duration. The night prior to admission the patient passed five to six tarry stools and, 2 hours before hospitalization, vomited coffee-ground material and lost consciousness. On admission he complained of dyspnea, nausea and midepigastric pain radiating through to the back. His past history revealed 10 years of peptic ulcer disease with one previous episode of upper gastrointestinal hemorrhage requiring transfusions. A recent upper gastrointestinal x-ray series demonstrated an active duodenal ulcer. He admitted heavy alcoholic intake.

Physical examination revealed a well developed and well nourished male in acute distress complaining of severe back pain. Blood pressure was 140/60, pulse 150, respiration 36 and temperature 37.2° C. The thorax moved symmetrically and auscultation disclosed bilateral expiratory wheezes and rhonchi. There was diffuse guarding over the abdomen with moderate epigastric tenderness and hyperactive bowel sounds. There was no rebound tenderness. Laboratory data: hematocrit 25%, white blood count 26,500/mm.^{3} with shift to the left. Admission chest x-ray film was normal. Another chest x-ray film 4 hours later revealed left hydropneumothorax. A chest tube was placed in the left pleural cavity recovering 925 cc. of sanguineous fluid. An esophagram with Hypaque was noncontributory. Three hours after ingestion of methylene blue, the dye appeared in the chest bottle.

The patient was operated upon 19 hours after admission. Left thoracotomy was performed and a 3-cm. long tear of the esophagus just above the esophagogastric junction was discovered. There was evidence of chemical and bacterial mediastinitis. The esophageal rent was closed with two layers of interrupted chromic catgut sutures and a large chest tube was connected to underwater drainage. On the eighth postoperative day an esophagopleural fistula became evident. On the ninth postoperative day, left empyema was drained with resection of two ribs and gastrostomy was performed. On the 16th day the patient underwent partial gastrectomy, gastroenterostomy and jejunostomy for massive bleeding from a large posterior duodenal ulcer.

His clinical course subsequently was one of gradual deterioration secondary to malnutrition and staphylococcal empyema with a persistent esophagopleurocutaneous fistula. He died 8 weeks following emetogenic rupture of his esophagus. Autopsy demonstrated an esophagopleurocutaneous fistula with a large empyema cavity, status post gastrectomy and marked wasting.

Case 2. A 42-year-old Puerto Rican man was admitted to Bellevue Hospital on September 5, 1963 with complaints of hematemesis and tarry stools of several hours duration. Following a bout of weekend drinking, and 8 hours prior to admission, he had an episode of painless vomiting of bright red blood. This recurred twice and was followed by two episodes of tarry stools. He had been in excellent health until 2 weeks prior to admission when he noted the onset of recurrent periumbilical pain relieved by food.

Physical examination revealed a well developed and well nourished alert male with blood pressure 90/50, pulse 124, respiration 17 and temperature 37.6° C. Chest was clear to percussion and auscultation. He had moderate epigastric guarding and tenderness without demonstrable rebound tenderness. Laboratory data: hematocrit 45%, white blood cell count 9,000/mm.^{3} with 82% polymorphonuclear leukocytes. Admission chest and abdominal x-rays were unremarkable. The patient was transfused with 1,500 cc. of whole blood. He continued to vomit blood around a nasogastric tube and 1 hour following admission he suddenly began to complain of intractable back pain. Blood pressure was 110/60, pulse 120 to 140. He had slight dyspnea with a respiratory rate of 28 to 32. His hematocrit was 44%. The electrocardiogram was unremarkable and portable chest x-ray film demonstrated a questionable widening of the mediastinum. By the sixth hour after admission his temperature had risen to 39.4° C.

He was operated upon 7 hours after admission through an upper midline abdominal incision. Through a gastrotomy it became evident that the bleeding was from the esophagus. The duodenum and stomach appeared normal. The index finger was passed into the esophagus and a rent along the left posterior aspect of the distal esophagus was felt. The esophagus was isolated below the diaphragm but the rent could not be brought down. The incision was extended into the left chest. There was approximately 1,000 cc. of blood in the left pleural cavity. The diaphragm was incised down to the esophageal hiatus. Just above the cardioesophageal junction there was a 3-cm. longitudinal laceration through all layers of the esophageal wall with actively bleeding edges. There was evidence of chemical mediastinitis and dissection of blood into the entire posterior mediastinum. Mucosal edges were visualized and closed with interrupted 4–0 silk and the muscularis was closed in similar fashion. This closure stopped the bleeding. The mediastinum was opened widely into the left pleural space and a large chest tube was placed. A gastrostomy was performed before closure of the abdomen. On the sixth postoperative day, 750 cc. of sanguineous fluid was aspirated from the *right* chest. Cultures grew no organisms and effusion did not recur. Three weeks postoperatively in the face of a persistent fever and with the later evidence of empyema of the left chest, segments of the ninth and tenth ribs were resected posteriorly for adequate drainage. Thereafter, the fever lysed and the empyema space slowly granulated in and healed.

The patieint was discharged 10 weeks following admission. At that time, and 6 months later, esophagograms and upper gastrointestinal x-ray series were normal.

TABLE 2. *Summary of Reported Cases*

Case	Age	Rupture Time to O.R.	Assoc. Disease	Complications	Course
1	46	21 hours	Duodenal ulcer with hematemesis	Empyema, left; esophagopleural fistula	Died in 8 weeks
2	42	6 hours	Mallory-Weiss syndrome with hematemesis	Empyema, left; pleural effusion, right	Discharged in 10 weeks

Discussion

Both patients (Table 2) represent instances of forceful emesis leading to rupture of the lower esophagus. In the first, we believe rupture occurred 2 hours prior to admission during a bout of hematemesis. The second patient presented with Mallory-Weiss syndrome but rupture of the esophagus probably did not occur until an hour after admission when, with continued vomiting, there was sudden onset of severe unremitting back pain and deterioration of the patient's clinical condition. Both patients presented with massive hematemesis, due in the first to a bleeding duodenal ulcer and in the second to Mallory-Weiss syndrome.

In each instance rupture of the esophagus was signalled by the abrupt onset of severe back pain. Although hematemesis need not accompany emetogenic rupture of the esophagus,[14, 22] vomiting and back pain are characteristic symptoms. The pain may simulate that of coronary thrombosis, dissecting aneurysm, pleurisy, perforated peptic ulcer or acute pancreatitis. Upper abdominal rigidity and tenderness in our two patients were secondary to pleural irritation and mediastinitis. Abdominal signs are confusing and tend to direct attention away from the primary disease.

Shock, which is usually present, may at first respond to treatment, but tachycardia is persistent and soon shock becomes intractable unless operative intervention corrects the underlying cause. Absence of shock obviously does not rule out the diagnosis of emetogenic rupture of esophagus.

Subcutaneous emphysema, particularly in the neck, is a sign of esophageal rupture into the mediastinum and was not a feature in either of our patients. This is a late and inconstant sign.[9, 18, 19, 22] On the other hand, copious left pleural effusion with or without pneumothorax usually follows esophageal rupture.

Chest x-ray studies are helpful if mediastinal emphysema or hydropneumothorax is present (Case 1). These findings are not constant and various series [9, 18, 19, 21, 22, 23] commonly included left hydropneumothorax or left pleural effusion, bilateral hydropneumothorax, bilateral pneumonia or normal pleural space. The changes may be subtle in early mediastinitis (Case 2). In both our patients, normal chest x-ray films immediately on admission, followed by the later development of abnormalities, indicate that changes are rapid and that serial chest x-rays are advisable. Early negative radiographic findings mean little.

Radiopaque materials and oral methylene blue to demonstrate esophageal rupture may confirm available evidence but frequently are not necessary for a correct diagnosis and may consume valuable time.

Successful treatment of emetogenic rupture of the esophagus is surgical and was accomplished only within the last 20 years. In 1900, Bowles [16] first recommended mediastinotomy with suture of the esophageal laceration. Foggitt,[11] in 1946, approached a

correctly diagnosed rupture of the esophagus transabdominally, pulled the esophagus below the diaphragm and sutured it. However, the patient died of mediastinitis. Transabdominal suture is not always possible (Case 2) and, in any case, drainage of the mediastinum, which is an essential part of therapy, cannot be performed adequately via the transabdominal route. In 1947, Barrett [1] and Olsen and Clagett [17] first reported successful treatment of esophageal rupture by left thoracotomy and suture of the perforation with mediastinal drainage. We believe that this is the correct approach; but effective surgical intervention must be prompt. In Case 1, although the diagnosis was suspected, surgical intervention was delayed until confirmation was obtained. We consider this an error in management.

Summary and Conclusion

The term emetogenic rupture of the esophagus is proposed in place of spontaneous rupture. The relationship of Mallory-Weiss syndrome to emetogenic rupture of esophagus is discussed and the two conditions are compared. Two cases of emetogenic rupture of the esophagus are reported. From these it is evident that 1) hematemesis may be part of the symptom complex, 2) the disease should be suspected in all cases of forceful emesis with severe acute back pain, with or without shock and 3) surgical intervention must be early if the high mortality and morbidity rate associated with this disease is to be improved.

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