Case Report Section

Spontaneous Rupture of the Esophagus

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Spontaneous rupture of the esophagus has attracted relatively little attention from the medical profession, and until recently, it has seldom been mentioned in the differential diagnosis of acute upper abdominal and chest pain.

The first recorded case was that of Boerhaave¹ in 1724. His patient, Baron Wassenaer, Grand Admiral of the Dutch Navy, frequently overindulged in eating and often induced vomiting in the Roman fashion. One day in 1723, after induced vomiting following a heavy meal, he was seized with agonizing chest pain which caused him to feel a bursting sensation in the chest and to collapse and roll about in agony, retching but not vomiting. For 18 hours, he was unrelieved of his suffering despite all efforts of his attending physician. Postmortem examination disclosed gastric content free in both pleural cavities due to rupture of the esophagus, a short distance above the diaphragm.

Until recently, such a catastrophe remained largely an autopsy room curiosity. Prior to 1946, Eliason and Welty² reported the mortality to be 100 per cent excluding two cases reported by Graham.

Early in 1946, Barrett³ made the correct diagnosis, performed thoracotomy, sutured the esophageal defect, and his patient recovered. Later in 1946, Olsen and Clagett⁴ were able to make the diagnosis immediately after rupture, and within three hours the defect was repaired trans-thoracically with success. To these men must be given great credit for their pioneering approach to this problem.

In a review of the literature in 1952, Ware, Schnider, and Davis⁵ collected a series of 86 cases that they felt fulfilled the criteria of spontaneous esophageal rupture. They were able to ascertain that 18 of these cases had recovered following surgery.

Definition: Spontaneous rupture of the esophagus refers to a normal esophagus rupturing from an excessive intraluminal pressure, generally induced by vomiting, and excluding those perforated by instrumentation, by ingested foreign bodies, or those due to malignant or benign ulcers. An esophagus that has been strictructed by esophagitis should not be included. One that is associated with a hiatal hernia, is likely afflicted with esophagitis. Likewise, the esophagus of an individual who has been retching for days, may well be eroded due to peptic ulceration.

Site of Rupture and Mechanism: Ware, et al⁶ found the left side of the esophagus torn in 39 cases, the posterior side in 11, and the right side in eight. The average length of the tear in the recorded cases has been 2.24 cm., with extremes from 0.6 cm. to 8.9 cm.
McKenzie\textsuperscript{a} in 1884, Burt\textsuperscript{7} in 1931, Hertzog\textsuperscript{a} in 1947, and Mackler\textsuperscript{a} in 1952 performed distention experiments on segments of the intestinal tract of cadavers. All authors found the stomach less able to withstand intraluminal pressure than the esophagus before rupturing. The esophagus could occasionally tolerate pressures up to seven pounds per square inch although it generally ruptured between three and four pounds pressure per square inch.

Some have denied the possibility of the normal esophagus rupturing from vomiting. They point out that many cases so reported have had repeated vomiting and probable peptic esophagitis. Some reports have included cases with stricture proximal to the rupture; other reports have been of congenitally short esophagus in conjunction with diaphragmatic hernia. A significant number of cases have had associated gastric and duodenal ulcers.

Because of experimental observations on the cadaver that the esophagus withstands pressure better than the stomach, these authors have concluded that spontaneous rupture from vomiting can never occur in an esophagus that has not been the site of weakening from previous disease.\textsuperscript{10}

On the other hand, most reported cases have no symptom to suggest previous esophageal disease, and clinically are spontaneous ruptures. Is it possible for humans to generate sufficient pressure to rupture the esophagus? Kinsella\textsuperscript{11} states that a well muscled individual can blow pressures of 3.44 to 3.63 pounds per square inch. No one knows just how much pressure is created in vomiting.

A consideration of the mechanism of vomiting discloses how the esophagus can be ruptured and the stomach left intact \textit{in vivo}, even though the stomach in the cadaver is less able to tolerate pressure without bursting, than is the esophagus.

The propulsive forces is supplied by a great increase in intraabdominal pressure. This latter is brought about by contraction of the abdominal wall muscles simultaneously with the diaphragm. At the same time, there is contraction in the region of the incisura and relaxation of the cardia and the esophagus. The intragastric pressure thus is increased largely due to compression from without and the uniform pressure surrounding the stomach prevents it from rupturing. The esophagus, in the thoracic cavity, a place of normally negative pressure, has no great positive pressure to surround it during emesis. If vomiting is too rapid to permit free egress of vomitus, or if any portion of the esophagus should go into physiological spasm during vomiting, that segment below it would be subjected to relatively high pressure.

Additional evidence of the spontaneity is afforded by reported cases of esophageal rupture occurring as a result of having been struck over the abdomen by a falling mantel,\textsuperscript{12} run across the abdomen by a motorcycle,\textsuperscript{13} or having been pinned beneath an automobile,\textsuperscript{14} and having the esophagus ruptured by compressed air.\textsuperscript{9}

\textit{Diagnosis:} Very little of significance in the clinical history has been added since the original description by Boerhaave.\textsuperscript{1} A close study of the
vast majority of the cases will reveal a similar clinical story. Usually the patient is a healthy man, often of stocky build, between 35 and 55 years of age. For any number of reasons, severe vomiting ensues, and at the height of a particularly strong emesis, the patient notes extreme upper abdominal and chest pain. The latter is most often noted on the left side, and may extend into the left shoulder. A sensation of “bursting something” in the chest is frequently noted. Shock, with clammy cold skin, rapid respirations, and cyanosis may promptly ensue. Emphysema of the cervical tissue may develop. Pain is unrelieved by the usual doses of narcotics. If nothing is done therapeutically, death almost invariably takes place within 48 hours, more often within the first 24 hours. Often, the diagnosis can be made from the clinical history.

This catastrophe is most frequently confused with perforated peptic ulcer of the stomach or duodenum, dissecting aortic aneurysm, acute hemorrhagic pancreatitis, coronary occlusion, and spontaneous pneumothorax.

In spontaneous pneumothorax, the accumulation of pleural fluid is slow and small in amount as contrasted to the accumulation of pleural fluid from irritation and spillage of gastric content from ruptured esophagus. Except for the occasional development of tension pneumothorax, the person with spontaneous pneumothorax is seldom very ill.

An upright or lateral decubitus roentgenogram of the abdomen will help rule out a perforated peptic ulcer and a normal serum amylase determination will generally exclude acute pancreatitis. An electrocardiogram will usually be abnormal if coronary occlusion is present.

The diagnosis of spontaneous esophageal rupture can be confirmed by the administration of lipiodol by mouth followed by a roentgenogram of the chest. If the esophagus be perforated, the dye will accumulate outside the esophagus, either in the mediastinum, or in the pleural cavity. Methylene blue in dilute solution may be administered by mouth and recovered by thoracentesis if the esophagus is ruptured. Often the pleural fluid will test acid to litmus paper due to the content of hydrochloric acid from the gastric juice.

Treatment: Antibiotics, blood transfusions, general supportive measures, oxygen therapy, and intratracheal anesthesia have all been important factors in survival and recovery. Treatment largely depends on the rapidity with which the diagnosis can be made. Sampson15 feels that it is the rare patient that survives long enough for infection to develop. He advocates early thoracotomy with repair of the linear rent as soon as possible. Certainly, this is the optimum treatment if the diagnosis has been made early.

The great majority of esophageal rents that have been repaired later than 12 hours after onset of the tear have subsequently broken down with development of empyema. On the other hand, there are a number of patients who have survived following rib resection and drainage of the pleural cavity. It has been necessary to employ rather rigorous supportive measures in such individuals but the esophageal rent has healed spontaneously in such cases.
It would seem advantageous to employ immediate thoracotomy and repair of the esophageal rent if the diagnosis is made early. If the diagnosis is not made prior to 12 hours after the accident, it would seem more prudent to employ drainage of the pleural cavity, either through intercostal tubes or by means of rib resection. Later, when the esophageal rent closes spontaneously, decortication and reexpansion of the lung may be carried out.

A useful procedure to tide such patients over the long stage of malnutrition while the esophageal rent is healing is the performance of jejunostomy as advocated by Lynch. Occasionally one is able to avoid jejunostomy by inserting a Miller-Abbott tube through the oral cavity, past the rent, into and through the stomach and duodenum, and permitting the tip to remain in the jejunum. One can then give jejunostomy feedings for an indefinite period of time.

Case Report

The patient was a white male, age 40, complaining of difficulty in breathing, and severe pain in the left chest and epigastrium. He had undergone repair of a right inguinal hernia on February 16, 1954 at the Adams County Hospital in Decatur, Indiana, and the operation had been carried out uneventfully under spinal anesthesia. Later that day, he had a few sips of water and tea, when suddenly he became nauseated and vomited in a most forceful fashion. At that moment, he seemed to twist his body and immediately, he noted excruciating left chest and upper abdominal pain. Breathing became labored and extremely painful. Cyanosis of the nail beds was evident. His past history was essentially negative and he had never experienced dyspepsia or dysphagia.

Examination disclosed an acutely ill male breathing with difficulty and slightly cyanotic. The blood pressure was 140/80 mm. Hg., the pulse 130, and respirations 40 per minute. The breath sounds were absent over the left side of the chest and there was hyperresonance to percussion over the entire left side of the thorax. The apical beat was shifted to the parasternal line on the left side. A diagnosis of spontaneous pneumothorax was considered and a needle was inserted into the left pleural space. Free air was obtained but his condition failed to improve.
In the subsequent two days, serum amylase determination was normal and an upright roentgenogram of the abdomen failed to disclose free air under the diaphragm. A film of the chest (Fig. 1) disclosed complete collapse of the left lung with a large amount of fluid in the left pleural cavity. Methylene blue solution was administered orally and was recovered from the left pleural cavity, thus proving the diagnosis of spontaneous esophageal rupture.

On February 19, 1953, he was transferred to the Ft. Wayne Lutheran Hospital (No. 53-1408). He was obviously quite ill and it was necessary for him to be in an oxygen tent because of the difficult and labored respirations and the cyanotic tinge to the nail beds. It was impossible to control the chest pain, despite the administration of large doses of morphine. Two intercostal catheters were inserted into the left pleural cavity on the day of admission. He was placed on intravenous fluids, blood transfusions were given, and huge doses of antibiotics were administered. Oral intake was discontinued.

Copious quantities of thick yellow pus continued to drain from the intercostal catheters and on February 26, 1953, another larger catheter was inserted into the left pleural cavity and connected to mild suction. The following day, a Miller-Abbott tube was placed through the mouth, esophagus, stomach, duodenum, and into the jejunum and a 4,000 calorie jejunostomy formula was administered daily thereafter until the esophagus healed. On March 5, 1953, under general anesthesia, a rib resection was carried out. The left lung was completely collapsed and covered with a tough rind. The problem of pleural drainage was thus solved and his condition began to improve rapidly.

By March 21, 1953, it no longer was possible to recover orally administered fluids from the left chest, and accordingly the Miller-Abbott tube was removed and liquids were administered by mouth. He was dismissed from the hospital.

Two weeks later he was readmitted and under general anesthesia, decortication of the left lung and chest wall was performed. The site of previous rib resection was closed as was the operative incision, and two catheters were placed in the pleural space and connected to negative pressure. Supportive therapy and large quantities of antibiotics were given. He made an uneventful recovery and was dismissed on April 26, 1953. The wounds were healed and the left lung was completely expanded.

He has returned to his regular occupation and his duties and abilities have not been limited. The latest chest x-ray films show an essentially normal chest except for the absent ribs at the site of operative interventions.

SUMMARY

A brief review of the pertinent history, site of rupture, mechanism of rupture, diagnosis, differential diagnosis, and therapy of spontaneous rupture of the esophageal is presented.

A case of spontaneous esophageal rupture successfully treated by drainage of the pleural cavity followed by decortication of the lung and chest wall is prescribed.

RESUMEN

Se presenta un resumen de la historia pertinente, el sitio de ruptura, el mecanismo de ésta, diagnóstico, la diferenciación, y el tratamiento de ruptura espontánea del esófago.

Se describe un caso de ruptura espontánea del esófago tratada con éxito por drenaje de la cavidad pleural seguido de decorticación del pulmón y de la pared de tórax.

RESUME

Les auteurs font une revue rapide de l'historique de la rupture spontanée de l'oesophage. Ils rappellent le siège de la rupture, son mécanisme, les éléments du diagnostic différentiel, et du traitement. Ils rapportent une observation de rupture spontanée de l'oesophage heureusement traitée par drainage de la cavité pleurale, suivi de décortication du poumon et de la paroi thoracique.
REFERENCES


Multiple Saccular Aneurysms of the Thoracic Aorta with Spontaneous Rupture into the Esophagus:
Report of a Case*

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Saccular aneurysm is "a pouching of the vessel at one point" (W. Boyd). Saccular aneurysms of the aorta are almost always syphilitic in origin. Because syphilitic aortitis usually develops in the thoracic aorta, it is the thoracic portion of this vessel which is especially affected. Syphilitic aneurysms of the abdominal aorta are relatively infrequent. Aneurysmal sacs may arise from the ascending, transverse, and descending segment of the arch, and, least frequently, from the descending aorta.

Multiple sacs may develop, though this is unusual. Of 633 cases of saccular aneurysms of the thoracic aorta, reported by Kampmeier, only 23 or 3.6 per cent were multiple.

Thoracic aneurysms may rupture into the surrounding structures including the pericardium, pleural cavity, esophagus, bronchus, etc. The present report is concerned with a case of multiple saccular aneurysms of the thoracic aorta, one of which perforated into the esophagus.

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