Esophagopleural Fistula after Pneumonectomy*

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Rupture of the esophagus into the space left after pneumonectomy is a rare and often fatal complication. Esophagopleural fistulas occurred in three patients following pneumonectomy. After previous methods failed, two patients were successfully treated by using a one-stage procedure which included (1) suture closure of the fistula, (2) buttressing the repair with a viable, pedicled, two-rib intercostal-muscle flap, and (3) performing an extensive thoracoplasty with a continuous drip infusion of neomycin. Such a procedure offers the maximum opportunity for successful treatment of this catastrophic lesion.

Rupture of the esophagus into the space left after pneumonectomy is a rare and frequently fatal complication. The three cases presented illustrate the difficulties in the management of this catastrophe. One patient died in the immediately postoperative period after a colon bypass procedure. The other two patients have remained asymptomatic following fistula closure by the procedure advocated by Engleman and co-workers.

CASE REPORTS

CASE 1

A 61-year-old man underwent pleuropneumonectomy in June 1968 for a destroyed right lung with empyema and persistently positive sputum cultures for acid-fast bacteria. During the difficult dissection, a small area of the middle portion of the esophagus was contused without mucosal perforation. The muscular wall was reinforced with sutures. Three weeks after surgery, the patient developed an esophagopleural fistula at the level of the repair. After initial drainage with a tube, a two-layer repair of the 1.5 × 1 cm perforation was performed with a gastrostomy and cervical loop esophagostomy. Within a few days the fistula recurred, and open-chest drainage was performed. The patient was maintained by feedings through the gastrostomy tube, with the saliva diverted through the cervical esophagostomy. The fistula failed to close. In April 1969, a substernal right colon-bypass procedure was performed. The patient died suddenly on the seventh postoperative day from a massive pulmonary embolus. Although oral feedings had not been started, the colon transplant and suture lines were intact.

CASE 2

A 51-year-old man was admitted in 1972 with a destroyed right lung. The sputum culture was negative for tuberculosis and fungi, but a variety of pathogenic organisms was cultured. Right pneumonectomy was performed. The esophagus was not adherent and was not injured. Cultures of the resected lung revealed Bacteroides organisms, and appropriate antibiotic therapy was administered. The patient did well until three weeks after surgery, when a small bronchopleural fistula developed, requiring drainage with a tube. Neither transbronchoscopic cauterization nor secondary closure with pleural flaps was successful in obliterating the fistula. Open drainage of the pleural space (Eloesser2 flap) was performed; the patient was subsequently discharged, and the fistula closed. Approximately one year later, after a night of "indigestion" and "retching," the patient noted that oral fluids came out of the pleural drainage site. A large misdesophageal fistula was demonstrated with a swallow of meglumine diatrizoate (Gastrografin®). Esophagoscopy and fiberoptic bronchoscopic examinations through the Eloesser flap showed a 1.5 cm defect. The right pleural space was reopened, and a portion of the dense mediastinal scar was divided to permit a two-layer closure of the esophageal perforation. A pedicled flap of the fourth and fifth intercostal bundles was sutured over the repaired esophagus. A nine-rib thoracoplasty and feeding jejunostomy were also performed.

After surgery the patient did well, except for development of duodenal obstruction apparently due to a superior mesenteric arterial syndrome accentuated by high placement of the jejunostomy tube. Intravenous hyperalimentation was added to provide additional caloric intake. Barium swallow showed an intact esophagus. One year later, the patient was eating and gaining weight.

CASE 3

A 62-year-old woman underwent left pneumonectomy in 1971 for a destroyed lung with hemoptysis due to tuberculosis. After surgery the patient developed a bronchopleural fistula and empyema requiring open thoracostomy (Eloesser1 flap). The open thoracostomy gradually closed over the next
year; however, the patient steadfastly refused to have the flap revised. Consequently, the bronchopleural fistula remained open. Two years later, oral fluids and food began passing through the thoracostomy. Barium swallow and esophagoscopy showed a 2 cm perforation of the esophagus at the level of the hilum, with no distal esophageal obstruction. Oral intake was stopped, and intravenous hyperalimentation was instituted. Two weeks later, left thoracotomy was performed. A small opening in the bronchial stump was sutured, and the esophagus was repaired with one layer of silk sutures. An eight-rib thoracoplasty obliterated the pleural space. Ten days later, the postoperative barium swallow showed a residual esophageal leak. At reoperation a wider exposure of the esophagus was obtained, and the perforation was closed in two layers. The repaired esophagus and the intact bronchial stump were covered with an intercostal-muscle pedicle. After surgery the patient continued to receive intravenous hyperalimentation for three weeks before resuming oral feedings. A barium swallow performed ten weeks after her second esophageal repair showed no evidence of a leak. The patient remains asymptomatic one year later.

**Discussion**

The most common cause of esophageal fistula after pneumonectomy is injury to the esophagus during the removal of adherent lung, pleura, or lymph nodes. In this event the fistula manifests itself early in the postoperative course, as in our first case. Although causes of late esophageal rupture are less obvious, chronic infection of the pleural space is one likely mechanism. In two of our cases, the esophagus ruptured more than 15 months after open-chest drainage had been instituted for a postoperative bronchopleural fistula with empyema. In the third case, chronic infection certainly seems culpable because the Eloesser flap had narrowed to the point that adequate drainage of the residual bronchopleural fistula was impossible. In the second case the esophagus apparently perforated after "retching," suggesting that the mechanism was similar to that of the Boerhaave syndrome, with the buttressing effect of the lung. The esophagus thus ruptured at the area of least resistance, the empty right pleural cavity.

Treatment of this condition is difficult, and procedures to close the fistula have a high rate of failure. Takaro et al. found that only 21 percent of their collected cases of esophageal fistulas were eventually cured, and the mortality was 51 percent. Immediate treatment consists of cessation of oral intake and drainage of the pleural cavity. Since the esophagus usually perforates freely into the pleural space, immediate thoracotomy with mediastinal drainage is not indicated. If the patient is in poor nutritional balance, correction by intravenous hyperalimentation may be required. While nonoperative measures alone are rarely successful, occasional closures of small fistulas have been reported with only nutritional support and drainage. Urschel and co-workers reported successful closure of several late fistulas using esophageal exclusion and diversion in continuity, in addition to drainage, nutritional support, and antibiotic therapy; however, none of their fistulas followed pneumonectomy, and all had the advantage of the buttressing lung. Direct suture closure has a high incidence of failure unless the suture line is covered with a flap of viable tissue. Benjamin et al. reported two cases of successful secondary repair using viable pleural flaps. Successful resection and end-to-end anastomosis of the esophagus after failure of direct closure has also been achieved. Esophageal bypass procedures with stomach, small bowel, or colon have occasionally been successful, but the mortality is high with these extensive procedures. Successful closure of the fistula after thoracoplasty alone has been reported.

In 1965, Bryant and Eiseman demonstrated the value of the intercostal pedicle as an adjunct in aiding esophageal repair in experimental animals. Engleman and associates subsequently performed a successful one-stage procedure for the closure of a large esophageal defect. The operative technique included suture closure of the fistula, covering this repair with a pedicled two-rib intercostal-muscle flap, and performing an extensive thoracoplasty with a continuous neomycin infusion of the pleural space. In our two successfully treated cases, we employed this technique after failure of direct suture closure. The recent report of another successfully treated case of esophageal rupture after pneumonectomy using an intercostal-muscle flap indicates the value of the procedure in the treatment of this unusual complication.

Adjunctive measures, such as intravenous hyperalimentation, which was used in our two successfully treated cases, allows the patient to achieve a state of anabolism and will undoubtedly improve the survival rates with this complication. If the perforation of the esophagus is operated early, before longstanding changes of inflammation and infection develop in the space left after pneumonectomy, then the addition of a thoracoplasty may not be necessary. Procedures such as that described by Clagett and Geraci may be successfully used; however, in the resections for tuberculosis where longstanding infection has occurred, we believe that thoracoplasty is essential. Nonetheless, the critical factor in the success of this excellent procedure is the careful approximation of a pedicled flap of two intercostal-muscle bundles with an intact blood supply over the repaired esophagus.

**References**

We employed systolic time intervals (STIs) in the study of beat-to-beat changes in left ventricular performance in a patient with electrical alternans. This technique is widely accepted as a reliable method of evaluation of cardiac function and has been used for the detection of phasic changes in cardiac performance.

**Case Report**

A 19-year-old girl had precordial pain accentuated with respiration, dyspnea, and evening temperature elevations. The blood pressure was 122/70 mm Hg; the jugular venous pressure was elevated 2.0 cm above the sternal angle at 45°. The cardiac impulse could not be palpated. The heart sounds were muffled; no friction rub or murmur was heard. On a chest x-ray film, gross cardiac enlargement was seen. On the electrocardiogram, there was definite electrical alternans (Fig 1).

The STIs were recorded on a multichannel recorder (Hewlett-Packard A4578). The paper speed was 100 mm/sec. As proposed by Weissler et al the following intervals were measured: total electromechanical systole (QS), left ventricular ejection time (LVET), pre-ejection period (PEP), and the ratio of PEP/LVET. Thirty consecutive heart beats were recorded. Inspiratory movements were marked on the chart paper by a manual marker.

The STIs following the larger QRS complexes were added as one group and compared to those of the beats following the smaller QRS complexes by use of Student's paired t-test. The QS2 and LVET intervals were significantly shorter in beats following the smaller QRS complexes, while the PEP was longer and the PEP/LVET ratio was larger (Fig 2 and Table 1). Respiratory motions did not seem to affect the LVET in a different manner from that found in normal subjects by Weissler et al. Paradoxical pulse was not seen at any time in the LVET or blood pressure, nor was pulsus alternans detected by palpation or measurement of blood pressure.

After removal of 400 ml of yellowish fluid, electrical alternans disappeared. The STI no longer showed differences

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**Alternation of Left Ventricular Performance with Electrical Alternans**

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Systolic time intervals (STIs) were measured in a patient with massive pericardial effusion and electrical alternans. The total electromechanical systole and the left ventricular ejection time (LVET) were significantly shorter in beats following the smaller QRS complexes, while the pre-ejection time (PEP) was longer and the ratio of PEP/LVET larger. This beat-to-beat difference in the STIs suggests that an alternation in left ventricular performance accompanied the electrical alternans. After evacuation of the pericardial fluid, electrical alternans and STI alternation disappeared.

It is currently believed that excessive rotation of the heart due to a great accumulation of pericardial fluid accounts for electrical alternans.

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**Figure 1.** Scalar ECG before fluid evacuation (double sensitivity). There is definite electrical alternans, best seen in leads 1, aVL, aVF, V1, and V4.