Spontaneous Perforation of an Oesophageal Diverticulum

Report of A Case With Survival*

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Spontaneous rupture of the oesophagus is a medical emergency the outcome of which is often fatal. Although considered rare, 200 cases were collected from the medical literature by Moynihan in 1952. However, the diagnosis of this condition is frequently overlooked or may be made in terminal states or post-mortem. Early detection with surgical repair may mean the difference between survival or death.

Perforation of an oesophageal diverticulum presents the same type of emergency. The present case of a perforation of an oesophageal diverticulum with subsequent development of an acute mediastinitis, a sinus tract leading to the left pleural cavity, and empyema presents several interesting points in differential diagnosis.

Case Report

E. A., a 43 year old white married executive, was well until 30 minutes before admission to the hospital December 3, 1954, following ingestion of a large dinner. Suddenly, he experienced an unbearable crushing substernal pain accompanied by nausea, vomiting, profuse perspiration, faintness, and marked shortness of breath. Examination at the emergency room revealed a well-developed and well-nourished man in shock, cold, clammy, temperature 96.8° F., weak pulse of 128 and blood pressure 84/60. Heart sounds were regular, but distant. Lung fields were clear to auscultation and percussion. The upper abdomen was semi-rigid and somewhat tender. Leuvened and nasal oxygen were administered and the condition improved. The pain persisted and was relieved by opiates.

Past history was non-revealing except for a four months' history of epigastric distress relieved by antacids. There had been no tarry stools.

Upon admission, ruptured peptic ulcer, acute myocardial infarction, acute pancreatitis or ruptured oesophagus were suspected. An upright, flat film of the abdomen revealed no free-air beneath the diaphragm. An x-ray film of the chest was normal. An electrocardiogram revealed an occasional auricular premature systole but was otherwise within normal limits. Initial blood studies revealed hemoglobin of 14.0 grams, packed cell volume 42 per cent, leukocyte count 15,100 with 94 per cent polymorphonuclear cells, and sedimentation rate 16 mm. On the following day, fasting blood sugar was 98 mgms. per cent, non-protein nitrogen 30 mgms. per cent, serum amylase 82 mgms. per cent, and serum bilirubin 0.2 mgms. per cent. Serology was negative.

He was dyspneic, experienced considerable tachycardia during the early days of his admission and was critically ill. Temperature was a septic type ranging from 100 to 105° F. daily with frequent chills. Repetition of the flat film of the abdomen was negative on the third day as was also a film of the chest. Lipiodol swallow was non-revealing. Serial electrocardiograms during a period of 30 days were normal. The leukocyte count ranged from 15,000 to 26,500 with a high polymorphonuclear leukocyte count. The sedimentation rate rose to 38 mm., serum amylase ranged 50 to 88 mgms. per cent, urinalysis was negative. Stools were negative for occult blood. A tuberculin test 1.1000 was negative.

On the seventh hospital day (December 8, 1954), he was found to have flatness to percussion over the lower left lung field and absent breath sounds (Figure 1). An x-ray film of the chest for the first time revealed a small left pleural effusion. Thoracentesis yielded 30 cc. of a thin, yellow purulent fluid which upon culture revealed

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Figure 1: December 8, 1954, x-ray film of chest, revealing a small left pleural effusion.—
Figure 2: January 3, 1955, gastro-intestinal series, showing a diverticulum of the distal end of the oesophagus and oesophageal hiatal hernia.
Hemolytic Staphylococcus albus. Sensitivity tests were positive for erythromycin. Culture of the fluid was negative for M. tuberculosis and guinea pig inoculation was negative at the end of six weeks. Sputum examination was negative for M. tuberculosis by Ziehl-Neelsen stain and culture. Blood cultures were negative.

The fluid in the left pleural cavity increased, but was gradually relieved by repeated small thoracenteses, but the fluid became so thick that this was no longer possible.

On December 15, 1954, thoracotomy was performed and a drainage tube inserted. There was slight evidence of a widening of the mediastinum on chest x-ray films at this time suggesting mediastinitis. During hospitalization, he received parenteral fluids, transfusions, penicillin, streptomycin, erythromycin, and streptokinase and streptodornase.

Improvemnt was progressive following thoracotomy and by January 3, 1955, it was thought safe to investigate roentgenologically the gall bladder and upper gastro-intestinal tract. X-ray inspection of the gall bladder revealed normal function without stones. The upper gastro-intestinal series revealed a constriction and a diverticulum of the lower end of the oesophagus, and a small oesophageal hiatal hernia. There was a collection of barium in a cavity adjacent to the lower end of the oesophagus just above the cardiac end of the stomach. At one time during the fluoroscopic examination a small trickle of barium seemed to extend into the apparent diverticulum, denoting a sinus tract just beneath the diaphragm and extending above the diaphragm into the left pleural cavity (Figures 2 and 3). No sign of gastric or duodenal ulcer was present.

On January 5, 1955, the packed cell volume was 44 per cent, leukocyte count 9400, serum amylase 84 mgms. per cent. Stool examination was negative for occult blood. Urinalysis was negative. X-ray film of the chest revealed thickened pleura in the left lower chest area, otherwise normal. Temperature was 98.4° F., pulse 80.

On February 5, 1956, roentgenologic examination of the chest and upper gastro-
intestinal series were normal except for demonstration of a small oesophageal hiatus hernia and a small diverticulum of the lower end of the oesophagus. The final diagnosis was: (1) Ruptured oesophageal diverticulum. (2) Empyema. (3) Acute mediastinitis.

DISCUSSION

This case typifies the sudden onset of spontaneous perforation of the oesophagus with substernal pain, nausea, vomiting, dyspnea, and shock. This was followed by reflex tenderness and distention of the upper abdomen. Only after five days were diagnostic confirmatory signs found.

The conditions most commonly considered in the differential diagnosis are spontaneous pneumothorax, perforated peptic ulcer, myocardial infarction, dissecting aortic aneurysm, acute pancreatitis, and acute oesophagitis. A swallow of 10-20 cc. of lipiodol may frequently reveal a rent in the oesophagus. The typical history such as in this case accompanied by mediastinal emphysema, or fluid in the left pleural cavity may be diagnostic. Recognition in a typical case is easy if the diagnosis is suspected.

Excessive vomiting or severe sneezing may precipitate the condition. Sudden increase in intra-abdominal or gastric pressure by ingestion of a large meal as in this case, excessive drinking or straining at defecation may be the etiology. Case reports have indicated that perforation may follow an epileptic fit, lifting a heavy weight, or direct blows to the abdomen. Rarely, the presence of peptic oesophagitis may lead to necrosis and leakage from the terminal oesophagus. This was experimentally demonstrated by Brackney and his coworkers.

BIBLIOGRAPHY


