Tension Pyopneumothorax*
Rare Presentation of Ruptured Barrett’s Esophagus

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Tension pneumothorax following esophageal rupture is very rare. We report a case in which a perforated Barrett’s esophagus rapidly developed a spontaneous tension pyopneumothorax. The mechanism for the tension remains obscure. Knowing that a tension pneumothorax can occur with esophageal rupture can help prevent misdiagnosis.

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Esophageal rupture is common and numerous causes have been reported. The classic radiographic findings, regardless of cause, include pleural effusion, pneumothorax, pneumomediastinum, and subcutaneous emphysema. However, review of the English language literature reveals only one prior case of a tension pneumothorax due to rupture of the esophagus.

We report what is, to our knowledge, the first case of a tension pyopneumothorax resulting from a ruptured Barrett’s esophageal ulcer and the second known case of a tension pneumothorax due to esophageal perforation.

CASE REPORT

A 77-year-old white man presented to the emergency room with a 3-day history of cough, right pleuritic chest pain and increasing shortness of breath and a 1-day history of severe right costovertebral pain. He had undergone two hiatal hernia operations and an abdominal aortic aneurysm repair and was receiving medication for peptic reflux. He was in moderate respiratory distress, and scattered rhonchi were heard in his lungs bilaterally. He had right costovertebral angle tenderness and a soft, nontender abdomen. The WBC count was 12,600/mm³. The admission chest radiograph (Fig 1) was initially interpreted as showing right pleural effusion, possible right lower lobe consolidation, and a midline mediastinum, leading to the clinical impression of right-sided pneumonia with a parapneumonic effusion.

An abdominal computed tomographic (CT) scan was ordered to evaluate his upper abdomen and aorta. A regimen of ceftriaxone was started for possible pneumonia. However, while awaiting the CT scan, he rapidly developed a marked increase in shortness of breath and a decrease in oxygen saturation. He was given furosemide and a bronchodilator and became sufficiently stable to undergo the abdominal CT scan 5 h after admission.

On the CT scan, an unexpected right hydropneumothorax was seen, and the study was therefore extended to include more of the chest. The mediastinum was shifted toward the left (Fig 2), indicating the presence of a right tension hydropneumothorax. No mediastinal air was seen.

A chest tube was placed on the right with an immediate rush of air and rapid drainage of approximately 1,000 ml of light brown fluid. A chest radiograph obtained after insertion of the chest tube showed the mediastinum to have returned to the midline. The initial analysis of the pleural fluid showed a pH of 6.98 and a glucose level of 18 mg/dl.

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FIGURE 1. Admission chest radiograph shows right pleural effusion and right lower lobe consolidation. Mediastinum is midline. Left eighth rib is surgically absent. Left lower lobe is hypoperfused.

In the ICU, the preliminary diagnoses were pneumonia and empyema, with a bronchopleural fistula as the cause for the tension pneumothorax. The patient required supplemental oxygen; his tachypnea gradually decreased, and he remained afebrile. In the first 48 h the chest tube drained over 2,500 ml of serosanguineous fluid, without an air leak.

In the absence of a definitive diagnosis, another chest CT scan was obtained, which showed a communication between the esophagus and the right pleural space (Fig 3). Complete analysis of the pleural fluid showed the following values: pH, 6.9; glucose, 18 mg/
of rupture.''

Perforation was identified. A distal esophagotomy with a thoracic esophagogastrotomy was performed. Histopathologic examination of the esophagus showed a Barrett's esophagus with extensive ulceration and perforation of a Barrett's ulcer. The patient slowly recovered and was eventually discharged.

**Discussion**

Iatrogenic causes are responsible for 48 to 68 percent of esophageal perforations, with endoscopy and dilation the most common. Other causes include external penetrating or blunt trauma, pressure necrosis or direct laceration by a foreign body, severe emesis, and rarely, perforation of a Barrett's ulcer or esophageal diverticulum.

Barrett's esophagus is generally an acquired lesion secondary to chronic gastroesophageal reflux with columnar epithelium found within the esophagus. Ulceration and strictures have been observed with Barrett's esophagus. The incidence of adenocarcinoma is said to be 10 to 41 percent. Of the hundreds of cases of Barrett's esophagus in the English language literature, we found only 12 cases of rupture. Perforations into the pleural space and into adjacent mediastinal structures have been reported. The site of perforation was not specified in two cases.

The common clinical findings with esophageal perforation from any cause are nonspecific and include chest and back pain, subcutaneous emphysema, fever, and dyspnea. The plain chest radiographs often show pneumomediastinum, pneumothorax, and pleural effusion, but were normal in 33 percent of patients with iatrogenic perforations and revealed "nondiagnostic abnormalities" in others.

Esophageal rupture is associated with a mortality of 18 to 44 percent. According to Rigg and Walker, regardless of cause, delayed recognition of esophageal perforation is the rule, the major reasons being wrong diagnosis, failure to recognize the significance of the brown pleural fluid, and lack of awareness of the problem. Diagnosis is delayed for more than 24 h in 50 to 77 percent of cases and the "classic" history and symptoms are often absent.

The most important study to obtain is an esophagram. This has been shown to be diagnostic in 86 to 93 percent of cases. If the diagnosis is still not clear, a chest CT scan may demonstrate pneumomediastinum, abscess cavities adjacent to the esophagus, or the actual perforation site. The pleural fluid of esophageal rupture is characteristically purulent with an elevated amylase level. The treatment of esophageal perforation can be nonoperative or surgical depending on the location and nature of the perforation and the clinical setting.

Pertinent to our case, three of the four prior cases of Barrett's ulcer rupture with an esophagopleural fistula had chest radiographs with hydropneumothoraces. None had a tension pneumothorax. The other reported case of a tension pneumothorax from an esophageal perforation was secondary to a fistula from an esophageal diverticulum to the pleural space.

The presence of tension associated with the hydropneumothorax in our case remains unexplained. We know of no discussions in the literature that elucidate a mechanism for its development. Perhaps our patient's previous hiatal hernia surgery and associated changes served to create a check-valve type of obstruction at the site of rupture and thus permitted the tension pneumothorax to develop.

In summary, esophageal perforation is very difficult to diagnose, and delay in treatment is common. The rare presence of a tension hydropneumothorax in esophageal perforation is especially misleading and is potentially a further cause for delayed diagnosis and treatment. In the case we present, the chest CT findings and increased amylase in the pleural fluid were diagnostic. Knowledge that an esophageal rupture can cause a tension hydropneumothorax may prevent delay in the diagnosis of such cases.

**References**

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Pneumomediastinum Caused by Subcutaneous Emphysema in the Shoulder*

A Rare Complication of Arthroscopy

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Severe subcutaneous emphysema following arthroscopy of the shoulder developed in a 62-year-old man. It subsequently evolved into pneumomediastinum with respiratory distress, but the patient recovered spontaneously. To our knowledge, this constitutes the first report of pneumomediastinum caused by subcutaneous emphysema in the shoulder. (Chest 1993; 103:1606-07)

Subcutaneous emphysema in the shoulder causing pneumomediastinum has not been described. I wish to report such a case, which occurred as a complication of arthroscopy of the shoulder.

CASE REPORT

A 62-year-old man underwent arthroscopy of the right shoulder for debridement of the glenoid labral tear. General anesthesia was induced, and the trachea was intubated easily on the first attempt with an 8.0-mm oral endotracheal tube. It was then connected to a volume-cycled ventilator with a tidal volume of 600 ml and a rate of 8/min. Peak inspiratory pressure was around 15 to 16 cm H₂O. The shoulder joint was distended with saline solution, of which the continuous inflow and outflow were regulated by an arthroscopy infusion pump. Surgery and anesthesia were eventful, but toward the end of the 2-hour surgery, swelling and crepitation of the right arm and shoulder were noted. These rapidly spread to the right side of the chest, the neck, and the face and later also involved the left side as well.

A chest radiograph (Fig 1) demonstrated subcutaneous and mediastinal emphysema. Two hours after completion of surgery, the patient developed a fever of 38.4°C. He felt slightly short of breath and complained of sore throat and substernal chest pain. Arterial blood gas measurements, while the patient was breathing oxygen 2 L/min by nasal cannula, revealed that the PaO₂ was 57 mm Hg, the PaCO₂ was 38 mm Hg, and the pH was 7.43. With increase of FIO₂ to 40 percent (by a Ventimask), the PaO₂ increased to 81 mm Hg. Fiberoptic bronchoscopy disclosed no evidence of pharyngeal, laryngeal, or tracheal injury.

Imipenem-clavulanate therapy was started intravenously. He continued to run a fever for the next 3 days and then defervesced. Blood and sputum cultures grew no microorganisms. Subsequent chest radiographs showed no development of pneumothorax. Both subcutaneous emphysema and pneumomediastinum started to decrease on the third postoperative day and completely resolved on day 7.

DISCUSSION

Air in the mediastinum can originate from five sites: the neck, the alveoli, the airways, the esophagus, and the abdominal cavity. In the case presented, there was no evidence of airway, esophageal, or abdominal injuries. The patient was not under prolonged or high positive pressure ventilation and therefore unlikely to have development of alveolar rupture. Had alveolar rupture occurred, giving rise to pneumomediastinum and subsequently subcutaneous emphysema, the latter would have been more symmetrical rather than commencing and remaining predominant around the right shoulder as occurred in this case. Thus, it appeared that the subcutaneous air in the neck, which originated from the right shoulder, was the most likely cause of this patient’s pneumomediastinum.

Subcutaneous emphysema in the shoulder causing pneumomediastinum is, however, rather unusual. Deep cervical fascia divides the soft-tissue structures of the neck into three distinct compartments: the previsceral space, the visceral

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![Figure 1. Anteroposterior radiograph of the chest revealing the pneumomediastinum (arrows) and radiolucent streaks of air in the subcutaneous tissue.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21671/ on 04/05/2017)