In the case reported herein a patient developed bilateral pneumonia and septic shock and subsequently developed bilateral effusions. Chest roentgenograms, computed tomograms, and findings from analysis of the fluid within the chest were consistent with typical empyemas. When surgical debridement was attempted, the effusions were found to be anatomically extrapleural. Symptomatic improvement was noted following debridement. (Chest 1989; 95:933-35)

**Bilateral Extrapleural Effusions Complicating Bilateral Pneumonia**

Robert M. Bogen, M.D.,† John Simon, M.D.;‡ and Dennis L. Buschman, M.D.§

Pleural effusions occur as a result of an abnormality of fluid transport between the parietal and visceral pleura. Pleural effusions complicate pneumonia in up to 40 percent of the cases.1 We report a case of bilateral effusions complicating an episode of bilateral pneumonia which occurred extrapleurally (ie, posterior to the parietal pleura, and within the endothoracic fascia).

**CASE REPORT**

A previously healthy 48-year-old white woman developed a dry cough and “swelling” in her throat. She had no fever, chills, or headaches and was given erythromycin and an intramuscular injection of dexamethasone (Decadron). Three days later, she was admitted to a hospital with the same symptoms, as well as lethargy, shortness of breath, and “spasms” in her neck and shoulders. Physical examination on admission revealed a pulse of 128 beats per minute and blood pressure of 60 mm Hg by palpation. Erythema was noted on her upper chest, and enlarged, tender lymph nodes were palpated in the neck. Auscultation of the chest revealed bibasilar rales and pleural friction rubs at both bases. The WBC was 9,600/cu mm, with 29 percent neutrophils, 33 percent band forms, and 37 percent lymphocytes. Serum electrolyte levels and findings from urinalysis were within normal limits. The chest roentgenogram on admission (Fig 1) revealed bilateral lower lobe alveolar infiltrates.

A flow-directed catheter was passed through the left subclavian vein and demonstrated a pulmonary capillary wedge pressure of 5 mm Hg. The patient was treated with volume replacement, dopamine, cephalixin, and gentamicin intravenously. Her blood pressure rapidly improved, and further clinical improvement was noted over several days. A chest roentgenogram obtained on the sixth day of hospitalization (Fig 2) revealed bilateral effusions. A thoracocentesis was performed on the left side and yielded 250 ml of a turbid yellow fluid with the following values: WBC, 22,500/cu mm; differential cell count; 53 percent neutrophils and 47 percent lymphocytes; RBC, 7,000/cu mm; glucose, 5 mg/dl; LDH, 960 IU/L; total protein, 3.9 g/dl; and pH 7.0. Serum values from that time were as follows: glucose, 97 mg/dl; LDH, 354 IU/L; and total protein, 5.7 g/dl. Culture of the fluid from thoracocentesis showed no growth, and cytologic examination revealed no abnormal cells. A culture of blood drawn on admission grew *Hemophilus influenza* in one bottle on the ninth day of hospitalization. Therapy

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†From the National Jewish Center for Immunology and Respiratory Medicine and the University of Colorado Health Sciences Center, Denver.
‡Assistant Professor of Medicine.
§Associate Clinical Professor of Surgery.
¶Assistant Professor of Radiology.
Reprint requests: Dr. Bogen, National Jewish Center, 1400 Jackson, Denver 80206

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Bilateral Extrapleural Effusions Complicating Pneumonia (Bogan, Simon, Buschman)

FIGURE 1. Portable chest x-ray film taken on first day of admission for placement of Swan-Ganz catheter shows bilateral lower lobe infiltrates, particularly in retrocardiac area with obscuring of left hemidiaphragm. There is no evidence of effusion on left side at this time. This portable x-ray film may show some blunting of right costophrenic angle, suggesting very early effusion.

with cephalexin was discontinued, and cefuroxime was begun. On the 13th day of hospitalization, the effusions were noted to be increasing, and a chest tube was placed on the left side. A chest roentgenogram following insertion of the chest tube demonstrated a decrease in the left-sided effusion. The patient was discharged after three weeks with persistent bilateral effusions.

The patient was referred to the National Jewish Center for Immunology and Respiratory Medicine for a second opinion because of the persistence of these bilateral effusions. The medical history was significant for a partial thyroidectomy in 1974, without complication. Physical examination of the chest revealed dullness to percussion bilaterally, approximately halfway up the thorax. Decreased breath sounds were noted over these areas, and bronchial breath sounds were noted above them. No rales, rhonchi, or wheezes were auscultated. Chest roentgenograms revealed bilateral effusions which were not mobile. Computed tomography of the chest confirmed the probable location of the fluid with some pleural thickening. Chest ultrasound revealed a few pockets of free fluid. A thoracentesis of the left yielded 5 ml of thick yellow fluid, with the following values: WBC, 400,000/cu mm, with 70 percent neutrophils and 30 percent lymphocytes; RBC, 35,000/cu mm; total protein, 4.9 g/dl; and LDH, 9,915 IU/L. Smears for bacteria and acid-fast bacilli were negative for organisms, and cytologic examination revealed no abnormal cells. Pulmonary function tests revealed a TLC of 3.41 L (73 percent of predicted), FEV1 of 1.19 L (42 percent of predicted), FVC of 1.55 L (44 percent of predicted), and a Dco/VA of 4.97 (86 percent of predicted).

It was believed that the patient's dyspnea was a result of the large space occupied by the effusions. Bilateral decortications as staged procedures were recommended. After general endotracheal anesthesia, a standard left posterolateral thoracotomy was done and the chest cavity was entered. Virtually no fluid was visualized within the pleural space, and the visceral and parietal pleura were free of any inflammatory peel. Fluctuation was noted below the diaphragmatic pleura. A needle was introduced, and dense yellow pus was encountered. The pleura was then excised, and 1,200 ml of thick cheesy material was unmasked; this was in the space between the parietal pleural and the diaphragmatic fascia and muscle, as if this represented a "hidden compartment." This space was opened between the parietal pleura and the diaphragmatic muscle and fascia, and the debris was drained and removed. After debridement, the lung expanded well to completely fill out the pleural space. The chest was then closed in the usual fashion using two chest tubes. Ten days later, surgery was performed on the right side in the same fashion, with exactly the same findings. Although PFTs were not performed after surgery, the patient felt immediate relief of symptoms. Culture of the debrided material revealed a peptostreptococcal species, with no other growth.

DISCUSSION

The pleural space normally remains free of fluid. The balance of hydrostatic and oncotic pressures favors influx of fluid from the parietal pleura to the visceral pleura and drainage into the visceral pleural lymphatic vessels. An alteration of hydrostatic pressure, oncotic pressure, or capillary permeability, or an interruption of the pleural lymphatic vessels may result in a pleural effusion. An inflammatory process such as pneumonia is thought to cause effusions either directly by increasing pleural capillary permeability, or indirectly from increased pleural space oncotic pressure as a result of protein leaking from these capillaries.

The parietal pleura is attached posteriorly to the subserous (visceral) fascia which is indistinguishable from the endothoracic fascia (Fig 3). The endothoracic fascia itself is bound
posteriorly by the prevertebral fascia, inferiorly by the transverse (endoabdominal) fascia, and superiorly by the scalene fascia, which forms the suspensory ligament of the pleura. The retropharyngeal lymphatic vessels lie within this space. Infections of these tissues are rare.

Older surgical literature suggests that an infectious process may progress caudally from the retropharyngeal space to the bifurcation of the trachea, where the space is obliterated by approximation of the pleurae. Our patient’s “empyema” was clearly within the endothoracic fascia and involved the parietal pleura, but completely spared the visceral pleura. We considered the following as mechanisms: The first possible mechanism was contiguous spread secondary to pneumonia. The bilateral pneumonia in this patient was likely due to infection from H influenzae, although this cannot be proven. We initially presumed that the pneumonia had caused bilateral pleural effusions. Although this was the apparent explanation early in the course of our patient, it is the most unlikely one in view of the findings at thoracotomy. Bearing in mind that normal flow of pleural fluid is from the parietal to the visceral pleura, it is difficult to explain how an effusion could be produced outside the parietal pleura as a result of bilateral pneumonia without producing any abnormality of the pleural space. A second possible mechanism was bacteremic seeding. Hemophilus influenzae was recovered from one culture of blood. It is unlikely that the endothoracic fascia became infected through this route because there was no evidence of other abscesses, septic emboli, or endocarditis. The endothoracic fascia has not been noted in the literature to have a predilection for infection by this route. A third possible mechanism was post-traumatic inoculation. Oleothoraces, formerly used in the treatment of tuberculosis, may become infected. One case of a post-traumatic extrapleural effusion has been reported, in which an extrapleural hemotorax occurred following an episode of chest trauma and subsequently became infected. A post-traumatic cause cannot be the explanation in our case, since the effusions occurred bilaterally, and the right side was not instrumented during the acute episode. A fourth possible mechanism was direct extension along the fascia. Pulmonary infections from Actinomyces and Nocardi species may involve the pleura and extrapleural space through direct extension. A case of extrapleural bacterial empyema has been reported following an episode of submandibular infection (Ludwig’s angina). Direct extension of the infection was documented by findings of pus in the pretracheal fascial plane during tracheostomy; the fluid collection was unilateral and over the superior lateral thorax.

Since the presenting symptoms in this case were mainly related to the neck and throat, we postulate that an infection which occurred near the neck (such as a retropharyngeal abscess) was the initial event, and that this infection extended directly into the endothoracic fascia. This could account for the presenting symptoms referable to the neck, the erythematous and edematous rash on the chest, and the extrapleural location of the abscess. The pneumonia which was apparent radiographically was probably secondary to aspiration (suggested by the Peptostreptococcus cultured from the debrided material) or hematogenous spread. A detailed examination of the oropharynx at the time of presentation might have revealed evidence for localized infection. Surgical treatment of this finding might have prevented the subsequent extension into the chest cavity.

Although this patient’s outcome was good, our case illustrates the importance of considering unusual locations of thoracic infection when evaluating effusions in the chest. This is particularly important if the patient’s presentation includes symptoms not commonly associated with a pneumonic process.

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