Bilateral pleural effusions usually have a common etiology and similar fluid characteristics. However, in a patient with bilateral effusions receiving immunotherapy, such an assumption may be hazardous. In these patients, it is suggested that bilateral diagnostic thoracocentesis be performed; it is particularly indicated in the presence of unexplained fever. The following is an illustrative case.

**CASE REPORT**

A 57-year-old man was admitted to the Wood Veterans Administration Center on July 8, 1977, with progressive breathing difficulty of two weeks' duration and increased cough, with mucopurulent sputum of one-week duration. He also experienced orthopnea and paroxysmal nocturnal dyspnea. He had no chills, chest pain or hemoptysis.

He had had a subtotal gastrectomy for peptic ulcer disease 14 years ago. In May of 1975, a diagnosis of class IV lymphoma with transformation to chronic lymphocytic leukemia was made and he was treated with various anti-leukemic drugs. In January, 1977, he developed perinasal cellulitis, and coagulase-positive *Staphylococcus aureus* was isolated from culture of the drainage. He was treated with methicillin sodium (duration and dosage unknown), and the infection subsided. In February, 1977, all medications were discontinued except prednisone, which was continued at a daily dosage of 10 mg.

In the ensuing three months, he experienced severe malaise and a 13.75 kg weight loss. Enlargement of lymph glands and increase in leukocyte count was observed and he was admitted to the same hospital on May 5, 1977. The leukocyte count was 93,700/cu mm, with 100 percent lymphocytes. Chest roentgenogram (Fig 1A) showed a right-sided pleural effusion, which was not present in earlier roentgenograms. Thoracocentesis revealed a serosanguineous fluid with a protein content of 4 gm percent, leukocyte count of 22,000 per cu mm (all lymphocytes). Gram stain of the pleural fluid and culture were negative. A pleural biopsy was not performed. Because of symptomatic improvement observed after digitals and furosemide (Lasix) administration and the characteristics (lymphocytic exudate) of the pleural fluid, cardiac failure and leukemia were considered the causes of the effusion. During his hospital stay he developed a lower lip abscess, which yielded coagulase-positive *S aureus*. A transient increase in temperature was noted, but blood cultures were negative. No antimicrobials were given and he was discharged on May 21. A chest roentgenogram taken two days before discharge showed bilateral effusions; however, diagnostic aspiration of the new left-sided effusion was not done.

On examination he was found to be tachypneic and in moderate respiratory distress. His pulse was 112 beats/min, blood pressure 138/72 mm Hg and temperature 37.7°C. Jugular venous distention, laterally displaced apical impulse, enlarged liver and spleen, pitting pedal edema and diminished breath sounds at both lung bases were noted. There were no skin abscesses and no cardiac murmurs. Chest roentgenogram (Fig 1B), taken on the day of admission, showed bilateral pleural effusions, more notable on the left. In addition there was a right mid-zone infiltrate. Blood studies revealed a hemoglobin of 9.1 gm/100 ml and a leukocyte count of 200,000/cu mm (98 percent lymphocytes). Treatment for cardiac failure was intensified. Because of persistent low-grade fever of 37.2° to 38.3° C, left thoracocentesis was done one week after admission, and 800 ml of

"Contarini's Condition:" Bilateral Pleural Effusions with Markedly Different Characteristics

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A leukemic patient presented with bilateral pleural effusions. The effusions were markedly different in their characteristics: left-sided staphylococcal empyema and right-sided lymphocytic effusion. To our knowledge, there are no cases of this type reported in the modern literature. The importance of bilateral diagnostic thoracocentesis in patients on immunosuppressant therapy is emphasized.

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yellow fluid was removed. The fluid had a protein content of 4.6 gm percent, dehydrogenase of 2,500 IU, and leukocyte count of 23,400/cu mm; 91 percent of the cells were segmented neutrophils. Pleural fluid culture yielded a pure heavy growth of coagulase-positive S. aureus. Blood cultures which were repeated four times were all negative. A closed drainage of the empyema with a chest tube was established and treatment with nafcillin sodium was started. Prior to antimicrobial therapy, right thoracocentesis was done to rule out the possibility of bilateral empyema. The pleural fluid from the right side of the chest had a protein content of 4.15 gm percent, lactic dehydrogenase of 223 IU, leukocyte count of 11,800/cu mm (97 percent lymphocytes) and cultures were negative for bacteria, fungi and acid-fast organisms. Diagnostic workup of the right mid-zone infiltrate included multiple examinations of sputa for fungi, acid-fast bacilli, cytology and bronchoscopy and bronchial brushings, all of which were negative. A transbronchial lung biopsy revealed only non-specific organizing pneumonitis.

DISCUSSION

In the literature of the 17th century, there is reference to the presence of effusions of differing character in bilateral pleural effusions (Velschius, 1667; cited by Jarcho). Francesco Contarini, the 95th Doge of Venice, died on Dec. 6, 1625, following a 5 month illness with fever, emaciation and cough. Shortly before his death he had orthopnea, foul-smelling sputum and cardiac arrhythmia. At autopsy the heart was very large and "the cavity of the chest contained about five pounds of watery matter. The flaccid right part of the lung appeared to be full of thick mucous fluid. The left had turned entirely into a whitish gore contained in the pulmonary tunic as if in a sac." It would be difficult to disagree with Jarcho’s retrospective diagnosis (3 centuries postmortem) that Contarini suffered from cardiac hypertrophy and, in addition, had both hydrothorax due to heart failure and either empyema or lung abscess.

Effusion due to congestive cardiac failure may be complicated by an infection. In his textbook of pulmonary diseases, Hinshaw lists "secondary infection of a serous pleural effusion" as one of the conditions leading to empyema. Spontaneous empyema in a cirrhotic patient has been reported. The portal of entry of infection in the pleural space can be pulmonary, mediastinal, subdiaphragmatic or by direct inoculation (trauma, iatrogenic or postoperative). The majority of cases of empyema result from extension of a pulmonary infection. In our case, the pathogenesis of the empyema is unclear, but can be speculated upon. It is possible that the bilateral effusions initially had a common etiology and subsequently the left-sided effusion became infected. There was no suggestion of mediastinal or subdiaphragmatic infection, and there was no history of trauma or previous left-sided thoracocentesis. Perhaps the best explanation in this patient receiving immunotherapy is gravitational flow of infected buccal secretions from the lip abscess down to the lung, resulting in pneumonia and subsequent empyema by direct extension. A hematogenous route of infection is less likely in view of the noninfected right-sided effusion, negative blood cultures and absence of metastatic abscesses.

REFERENCES