intubation. The material obtained was examined by direct immunofluorescence with antiserum specific for the bacillus causing Legionnaires’ disease.

The specimen contained numerous small, rod-shaped fluorescent organisms. Therapy with erythromycin was begun, and within 12 hours the patient became afebrile, more alert, and responsive. During the following two weeks, there was almost complete clearing noted on the chest roentgenogram. The patient was extubated and was ambulatory in the hospital. Plans for discharge were being made when aspiration occurred following a large meal; the patient died a few hours later. Although no bacilli of Legionnaires’ disease were demonstrated in pulmonary tissue obtained at autopsy using cultures and direct immunofluorescent techniques, as well as transmission electron-microscopic examination, the serum titer of antibody against the bacillus of Legionnaires’ disease had risen from 1:28 on admission to 1:512 two weeks later.

DISCUSSION

Many have recognized the need for a more rapid diagnosis of this often serious and sometimes fatal disease. By employing the relatively safe technique of pulmonary aspiration via a needle, a rapid and reliable diagnosis can be made in some patients; in these instances the administration of unnecessary antibiotics is avoided. Normal findings on aspiration do not exclude the diagnosis of Legionnaires’ disease, since errors in sampling or technique may be expected on occasion. All samples from aspiration must be processed as for the diagnosis of any perplexing pneumonia.

We know of only one patient in whom immunofluorescent studies of a parenchymal pulmonary specimen obtained before death showed the bacillus of Legionnaires’ disease; the specimen was from pulmonary biopsy (type not reported). We believe aspiration via a needle to be preferable to pulmonary biopsy as an initial step in most cases. There have been several abnormal findings on direct immunofluorescent preparations of pleural fluid; however, pleural effusion only occurs in approximately half of the patients.

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REFERENCES


Hemoptysis in Patients with Septic Pulmonary Infarcts from Tricuspid Endocarditis*

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Three drug addicts with tricuspid valve endocarditis and pulmonary cavitation secondary to septic pulmonary infarcts developed massive hemoptysis. Two patients died of asphyxia and one survived. Both who died were improving clinically and had negative blood cultures when the fatal hemoptysis occurred. Therefore, however slight it may be, hemoptysis occurring in association with septic pulmonary infarcts associated with endocarditis may require prompt and aggressive measures.

The complications of infective endocarditis are well known. Congestive heart failure is the most frequent cause of death in endocarditis. Arterial septic emboli, such as coronary and cerebral emboli and mycotic aneurysms, are the second most common. Recently we have seen three patients with tricuspid valve endocarditis and septic pulmonary emboli, of whom two died of massive hemoptysis while their infection was under control. Mortality in endocarditis due to massive hemoptysis is rare. Between 1973 and 1978 we treated over 60 patients with tricuspid valve endocarditis and septic pulmonary emboli; hemoptysis occurred in only these three, and two of them asphyxiated in their own blood.

CASE REPORTS

CASE 1

A 22-year-old man was admitted because of fever, chills and cough. He had a long history of intravenous heroin usage. On physical examination, he was found to have an ejection systolic murmur at the left lower sternal border. Chest roentgenogram showed a dense infiltrate in the right lower lobe. Three blood cultures grew multiple organisms: Staphylococcus aureus, Bacteroides fragilis, Fusobacterium nucleatum, and Veillonella. After obtaining informed consent, the patient was started on intravenous cefoxitin 8 grams per day. Cefoxitin, a cephalosporin derivative, is active in vitro against S aureus and most anaerobic bacteria, including B fragilis. Sixteen days later, the blood cultures showed no organisms and the patient became afebrile and clinically appeared much improved. On the 17th hospital day, he

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noticed mild hemoptysis (a few streaks of blood in the sputum). The chest roentgenogram obtained at this time showed cavitation in the area of previously noted consolidation. Results of coagulation studies, including prothrombin time, partial thromboplastin time and platelet count, were normal. The next day the patient had a sudden episode of massive hemoptysis and asphyxiated despite resuscitation attempts. Autopsy performed by the coroner revealed vegetation on the tricuspid valve. In addition, there was a 5 cm abscess cavity in the right lower lobe of the lung filled with a large blood clot. Unfortunately, no cultures were obtained at autopsy. There was no evidence of an intra-abdominal infection.

**Case 2**

A 30-year-old man addicted to heroin was admitted with cough, fever and chills. The chest roentgenogram showed a density in the upper lobe of the lung. Three blood cultures grew alpha-hemolytic Streptococcus. He was begun on intravenous penicillin G, 20 million units per day. Five days later, the blood cultures showed no organisms, but ten days later, the chest roentgenogram showed a cavity with a fluid level in the area of previously noted consolidation. He was suspected to have an anaerobic lung abscess and therefore was switched to carbenicillin, 24 grams per day, on the 22nd day. On the 25th day, he noted mild hemoptysis. He was examined and found to have no evidence of a coagulation defect; bleeding time was normal. The next day he experienced massive hemoptysis and died despite attempts at resuscitation. Autopsy performed by the coroner’s office revealed vegetations on the tricuspid valve. The pulmonary cavity contained blood clots. No cultures were obtained at autopsy.

**Case 3**

A 22-year-old male heroin addict was admitted with cough and fever. A tricuspid systolic murmur was heard on physical examination. Chest roentgenogram showed cavitation in the center of the right lower lobe consolidation. Blood cultures grew alpha- and beta-hemolytic Streptococcus susceptible to penicillin at 0.1, cefotaxin at 0.5 and clindamycin at 2 μg/ml. He was treated successively with penicillin, 20 million units per day for two weeks; cefotaxin, 12 grams per day for two days; and then clindamycin, 2.4 grams per day for three weeks because of refractory bacteremia from the alpha-hemolytic Streptococcus, despite its being susceptible in vitro to all three antibiotics employed. During his entire course, he had intermittent hemoptysis. He was observed closely, but massive hemoptysis never occurred. Because of persistently positive results of blood cultures, he underwent surgical resection of the tricuspid valve. The tricuspid valve showed massive vegetations, but they were sterile on culture. After surgery, the hemoptysis resolved and the pulmonary cavity closed after several weeks.

**Discussion**

Endocarditis among drug addicts is on the increase. Most patients have tricuspid valve involvement. Septic pulmonary emboli result from the dislodgment of the vegetations, leading to necrosis and cavitation of the lung with abscess formation. That such cavities are due to infection is borne out by the fact that the causative organism has often been cultured from the sputum obtained by transtracheal aspiration. Pulmonary infection is thus the most frequent presentation of patients with endocarditis of the tricuspid valve. The cardiac murmur may be totally absent. Massive hemoptysis is defined as a single expectoration of more than 500 ml of blood. Hemoptysis has been reported as a complication of other disease entities, such as tuberculosis, bronchogenic carcinoma, bronchiectasis and primary lung abscess; however, it has not previously been reported as a fatal complication of septic embolization in tricuspid endocarditis. When massive hemoptysis occurs in such patients, they may suffocate rapidly. Localization of the bleeding segment by bronchoscopic examination is usually the first step in therapy; if active bleeding is seen, emergency thoracotomy is indicated for resection of the bleeding segment.

Somewhat alarming is the rapidity with which these patients develop massive hemoptysis. Within 24 hours of onset, what seemed like mild hemoptysis developed into an episode of massive hemoptysis that literally asphyxiated two of our patients. None of the three patients showed any evidence of a coagulation defect. When a patient with endocarditis with pulmonary cavi
tation secondary to septic emboli develops even mild hemoptysis, it is important to quickly localize the bleeding segment by bronchoscopic examination and watch expectantly for more severe hemoptysis. Bed rest is indicated. If severe cough is present, cough suppressants should be used. Emergency equipment for intuba
tion and suction should be kept at the bedside. Several units of blood should be kept on standby. If massive hemoptysis occurs, immediate intubation to clear the airway should be performed and thoracotomy to resect the bleeding segment must be considered promptly. Thoracotomy is indicated if an airway cannot be maintained due to continued bleeding or if blood pressure cannot be maintained despite adequate support of blood volume.

**References**


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