Tamponade Due to Rhodococcus equi in Acquired Immunodeficiency Syndrome*

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Infection with Rhodococcus equi has been described as a cause of cavitary pneumonia in AIDS patients. We report such a case, complicated by bacteremia, tamponade, and possible brain and liver abscesses. Medical treatment was successful with pericardiocentesis and antibiotherapy with teicoplanin, gentamicin, clarythromycin, rifampicin, supplemented by empirical treatment of cerebral toxoplasmosis. Antibiotherapy was terminated after 6 months, without relapse 2 months later. (Chest 1994; 106:1278-79)

Key words: acquired immunodeficiency syndrome; pneumonia; Rhodococcus equi; tamponade

Pneumonia caused by Rhodococcus equi, an aerobic Gram-positive bacillus, is currently well-known in patients with AIDS. Clinical illness usually is manifested as cavitary pneumonia. Disseminated infections have been described with bacteremia, brain abscesses, lymph node infection, diarrhea, pelvic abscess, and pleural infection, but symptomatic purulent pericarditis is unusual. We report such a case of an AIDS patient with disseminated infection and tamponade treated successfully by pericardiocentesis and antibiotherapy.

CASE REPORT

A 29-year-old AIDS patient was admitted to the Medical ICU because of pneumonia complicated by tamponade. He had a medical history of intravenous drug abuse and he was known as human immunodeficiency virus-1-positive since 1990. In October, 1992, CD4+ lymphocyte count was 80/mm³. He was hospitalized in February 1990 for hepatitis B and delta and in May 1990 for pneumococcal pneumonia. The patient had had fever (40°C), dyspnea, and cough with brown sputum for 3 weeks before admission. He was admitted to the Infectious Diseases Unit in November, 1992. A chest roentgenogram revealed pneumonia in the upper right lung and cardiac enlargement.

The leukocyte count was 7.3 10⁹/L; hemoglobin level, 82 g/L; hematocrit, 25 percent; platelet count, 376 10⁹/L. Arterial blood gas values were as follows: pH, 7.50; PaCO₂, 26 mm Hg; PaO₂, 69 mm Hg; HCO₃⁻, 21 mmol/L; oxygen saturation, 95 percent. The patient was admitted to the Medical ICU 4 days later because of respiratory failure and shock despite therapy with intravenously administered ceftriaxone, 2 g/d. On admission, blood pressure was 120/60 mm Hg; pulse, 130 beats per minute; temperature, 38.5°C; and respiration rate, 22 breaths per minute. Physical examination revealed paradoxical pulse, right heart failure, pericardial friction rub, bilateral rales, and multiple enlarged lymph nodes. A chest roentgenogram showed pneumonia of the right upper and the middle lobes, bilateral pleural effusion, and considerable cardiac enlargement (Fig 1). The ECG was compatible with pericardial effusion (tachycardia, T wave inversion, electrical alternans, low QRS complex voltage). Diagnosis was confirmed by echocardiography, revealing abundant circumferential pericardial effusion with signs of compression. Pericardiocentesis was performed under echocardiographic control, removing 1,800 ml of serohematic fluid (normal WBC-RBC ratio) with protein, 44 g/L. Bacteriologic samples were positive (blood cultures, bronchoalveolar fluid, pericardial fluid) for R equi. This strain was sensitive to aminoglycosides, erythromycin, rifampicin, glycopolyptides, and imipenem. The computed tomographic scan of the chest revealed cavitary pneumonia of the right upper lobe, pneumonia mass of the middle lobe, and mediastinal lymph nodes. The patient improved after pericardiocentesis and therapy with imipenem, gentamicin, vancomycin, and rifampicin. Because of persistent fever, a computed tomographic scan of the head was performed, revealing right cerebellous and left lenticular hypodense, ring-enhancing lesions with surrounding edema compatible with abscesses. Cerebral toxoplasmosis was suspected, and treatment was supplemented with sulfadiazine and pyrimethamine. A computed tomographic scan of the liver revealed three hypodense, ring-enhancing lesions (puncture was sterile). No exposure to farm animals was found. The patient left the hospital in January 1993 under a regimen of therapy including teicoplanin, clarithromycin, rifampicin, sulfadiazine, and pyrimethamine. In March, teicoplanin therapy was stopped, clarithromycin in April (by the patient), and rifampicin in May because of the clinical improvement. No relapse had occurred by July, 2 months after ceasing antibiotic treatment.

DISCUSSION

Pericardial effusions are detected by echocardiography in 10 percent of AIDS patients. Symptomatic pericardial
effusion and tamponade are more rare. They usually are due to Mycobacterium tuberculosis, opportunistic infections, Kaposi’s sarcoma, or other neoplasms. Among the infections, R equi is a rare pathogen, revealed in this case by pericardiocentesis. The low immune status of our patient and the delayed treatment probably explained the disseminated infection with bacteremia, possible multiple abscesses, and tamponade. We did not prove R equi infection in the liver and brain abscesses, but liver aspiration was performed 1 month after beginning antibiotic therapy. Brain biopsy was not possible, explaining why we treated the patient empirically for possible cerebral toxoplasmosis. Outcome of R equi infection is characterized by a mortality around 50 percent. Treatment is not well defined, but a combination of antibiotics is recommended to avoid frequent selection of resistant mutants. Treatment also is recommended for at least 2 to 6 months, perhaps for life. Several cases have been described with surgical treatment (lobectomy) for persistent abscess formation to supplement the medical treatment. In this case, pericardiocentesis permitted the removal of the infected fluid, proving the pericardial infection. No recurrence occurred and no surgical drainage was performed. The antibiotic therapy was extensive and protracted because of the resistance of the strain and because of possible cerebral abscesses. This case report describes a severe R equi infection with bacteremia, cavitary pneumonia, complicated by tamponade and possible brain and liver abscesses, with successful medical treatment. This type of infection should be suspected in an AIDS patient when pericardial effusion occurs with cavitary pneumonia.

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Ulcerative Tracheobronchitis Years After Colectomy for Ulcerative Colitis*


Two men with severe ulcerative colitis developed ulcerative tracheobronchitis 4 and 8 years after total colectomy. Intense plasma cell infiltration of tracheal mucosa and submucosa and destruction of mucous glands occurred, with partial relief of symptoms with corticosteroids. We compare them with the only other case reported, also years after colectomy.

(CHEST 1994; 106:1279-81)

Ulcerative colitis has a rare association with bronchitis, bronchiectasis, and bronchiolitis. To our knowledge, tracheal inflammation and ulceration have been described only in one patient, 14 years after colectomy. We describe two more patients with ulcerative tracheobronchitis that developed 4 years and 8 years after total colectomy for ulcerative colitis, and who responded to treatment with corticosteroids.

CASE REPORTS

CASE 1

A blacksmith and builder developed ulcerative colitis at age 28 years. He was treated with courses of salazopyrine and prednisolone until a polypoid adenocarcinoma was discovered at the age of 42 years and was removed by panproctocolectomy. Salazopyrin and prednisolone therapy was then stopped. At 44 years he developed a slight wheeze, and was treated intermittently with salbutamol, ipratropium bromide, and beclomethasone. He had not smoked since age 38 years. At 54 years he was referred with a remarkable very severe resonant deep-toned cough. Examination and routine investigations revealed only a slight wheeze and modest airways obstruction. Chest radiograph was normal. Rigid bronchoscopy showed thickened velvety irregular mucosa of trachea and main bronchi that bled easily on contact. The posterior wall of the trachea was floppy with pale nodules down the center. Deep biopsy specimens taken from the trachea and both main bronchi showed squamous metaplasia with intense plasma cell infiltration.

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FIGURE 1. Tracheal biopsy specimen of case 1 showing deep intense plasma cell infiltration. This biopsy specimen was not immediately beside an ulcer.