described in the cases presented by Cohen et al. and Anderson et al. Pericarditis due to MAI should be considered in the differential diagnosis of patients with AIDS who present with evidence of cardiac dysfunction, especially in patients with known MAI infection elsewhere. Given that the number of patients with AIDS is increasing, even rare infectious complications, such as pericarditis due to MAI, will be seen more frequently. If AFB are identified in pericardial fluid, therapy with multiple antimycobacterial agents should be initiated prior to bacteriologic confirmation. This therapy could control infection with M. tuberculosis; and while not necessarily effective in establishing a bacteriologic cure for MAI infections, the treatment might serve a suppressive role and thus prolong the patient's life. While MAI may not always be a direct cause of death, cases such as the present report confirm that MAI can certainly be lethal. Identification of MAI in patients with AIDS strongly deserves consideration for therapy.

ACKNOWLEDGMENT: We thank Ms. Michelle Fisher for her secretarial assistance.

REFERENCES


Pleural Effusion in Churg-Strauss Syndrome*

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A 33-year-old man with a two-year history of asthma and sinusitis presented with wheezing, pleuritis, bilateral pleural effusions, and patchy basilar infiltrates on chest roentgenogram. Laboratory studies revealed peripheral blood eosinophilia, and pulmonary function studies showed an obstructive pattern which was bronchodilator responsive. Thoracoctesis yielded an acidic exudative effusion with low glucose, low C3, eosinophilia, and a markedly increased rheumatoid factor. Open lung biopsy revealed extensive eosinophilic interstitial pneumonitis with nectrocytosing eosinophilic vasculitis. Although pleural effusions are present in 29 percent of Churg-Strauss patients, these effusions have not been well described. This report describes the pleural fluid findings in a case of Churg-Strauss syndrome.

(Chest 1989; 95:1357-59)

Churg-Strauss syndrome is a disorder of hypereosinophilia and systemic vasculitis in subjects with asthma and allergic rhinitis. Pleural effusions are commonly reported as a manifestation of this syndrome; however, the cellular and biochemical characteristics have not been well described. In this case of Churg-Strauss syndrome, the patient's pleural effusion is fully described, and the differential diagnosis of acidic eosinophilic pleural effusions is reviewed.

CASE REPORT

A 33-year-old nonsmoking man presented with a two-year history of sinusitis and progressively worsening asthma. Two weeks before admission, he developed pleurisy, first on the right and then on the left side of his chest, and a chest roentgenogram showed bilateral

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This study was supported in part by training grant HL07085 from the National Institutes of Health.

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pleural effusions. His peripheral WBC was 34,000 cells/cu mm with 66 percent eosinophils. A thoracentesis on the right yielded cloudy fluid with the following findings: LDH, 2,856 IU/L; protein, 4.2 g/dL; pH, 7.08; glucose, less than 10 mg/dL; amylase, less than 30 U/L; RBC, 600 cells/cu mm, and WBC, 10,400 cells/cu mm with a 95 percent eosinophils. Cultures for bacteria and mycobacteria were negative, and cytology was negative for malignancy. The patient was referred to National Jewish Center for Immunology and Respiratory Medicine for further evaluation.

Physical examination was unremarkable except for the lung exam, which revealed dullness at both bases and diffuse expiratory wheezing. The patient was afebrile. Blood studies revealed WBC 22,500 cells/cu mm with 62.8 percent eosinophils. Total eosinophil count was 14,130 cells/cu mm. Serum rheumatoid factor was 1:5120, serum antinuclear antibody was positive at 1:40, CH50 was 34 U/ml (normal 22-43), and C3, 113 mg/dl (normal 83 to 77). Immediate and delayed Aspergillus skin tests were negative. The PPD was negative. Examination of stool for ova and parasites was negative, and filaria and paragonia serologies were negative. Pulmonary function tests revealed airflow limitation which improved after inhaled bronchodilator therapy. The chest x-ray film revealed bilateral pleural effusions with patchy basilar infiltrates (Fig 1).

A left thoracentesis with Cope needle pleural biopsy revealed the following: clear yellow fluid; pH, 7.28; LDH, 1714 IU/L; protein, 6.5 g/dL; glucose, 6 mg/dL; cholesterol, 98 mg/dL; ANA, negative; rheumatoid factor, 1:10240; C4, 25.5 mg/dL; WBCs, 4290 cells/µl; eosinophils, 2,394 cells/cu mm; and RBCs, 5250 cells/cu mm. Pleural fluid cytology was negative for malignant cells. Pleural biopsy showed chronic pleuritis with eosinophilic infiltration. Bronchoalveolar lavage yielded 1.28 x 106 WBC/ml with 89 percent eosinophils, 3 percent lymphocytes, and 7 percent macrophages. Open lung biopsy of the right lower lung revealed patchy interstitial and intraalveolar inflammatory infiltrates rich in eosinophils, with prominent cuffs of inflammatory cells about small pulmonary vessels (Fig 2). Subpleural and interlobular connective tissue was heavily involved with eosinophil-rich inflammatory infiltrate, and dilated lymphatic channels were present in these structures (Fig 3). There was hypertrophy of bronchiolar musculature, bronchial basement membrane thickening, and reduplication of bronchiolar mucosa with goblet cell hyperplasia. Occasional arteries contained segmental or circumferential transmural inflammatory infiltrates with associated necrosis of a component of the vessel wall; mural macrophage giant cells were often present in these lesions (Fig 4). These histologic findings confirmed the clinical diagnosis of Churg-Strauss syndrome. Prednisone, 60 mg/day was started, and within
two weeks, the asthma symptoms, eosinophilia, and roentgenographic abnormalities resolved. The patient has been followed for ten months on tapering doses of prednisone without recurrence of his symptoms.

**DISCUSSION**

Although pleural effusions are present in 29 percent of Churg-Strauss patients, these effusions tend to be small and manifest only as occasional pleurisy. There are only two reported cases in which pleural fluid has been examined in Churg-Strauss syndrome, and neither of these reports comment on pleural fluid pH or chemistries. In this case of Churg-Strauss syndrome, two separate thoracocenteses show these effusions to be acidic exudates with marked eosinophilia and markedly low glucose.

The differential diagnosis of acidosic pleural effusions with low glucose is limited to esophageal rupture, infection, malignancy, and rheumatoid effusions. In this case, these causes were excluded by the results of pleural and open lung biopsies and by negative cultures of pleural fluid, pleura, and lung tissue. Rheumatoid effusions may rarely present before manifestations of joint disease, and rheumatoid arthritis patients may have peripheral eosinophilia. Also, there are case reports of eosinophilic pleural effusions in rheumatoid disease. However, these patients had between 2 and 31 percent eosinophils in their pleural fluid. In addition, all of these patients had rheumatoid skin nodules and most had joint disease and rheumatoid pleural nodules. The patient we describe lacked any joint, skin, pleural, or lung parenchymal evidence of rheumatoid arthritis. The increased rheumatoid factor in our patient is nonspecific and has been described in 52 percent of patients with Churg-Strauss syndrome.

There have been several reviews of eosinophilic pleural effusions. Most reviews stress that eosinophilic effusions are rarely associated with malignancy and usually indicate a "benign" course. Conspicuously absent from all lists is Churg-Strauss syndrome, despite the common occurrence of effusions with this disease. This case describes the cellular and biochemical characteristics of the pleural effusions in a case of Churg-Strauss syndrome. The differential diagnosis of acidosic exudative pleural effusions with low glucose should include Churg-Strauss syndrome.

**ACKNOWLEDGMENTS:** The authors thank Dr. Talmadge E. King for the bronchoalveolar lavage analysis and for assistance with this paper.

**REFERENCES**


**Pericardial Effusion and Tamponade due to Kaposi's Sarcoma in Acquired Immunodeficiency Syndrome**

*Jennifer L. Siotka, M.D.; Chester B. Good, M.D.; William R. Douen, M.D.; and Wahns N. Kapoor, M.D.*

We describe a 29-year-old homosexual man with acquired immunodeficiency syndrome who developed pericardial effusion and tamponade. Pericardioentesis resulted in clinical improvement. All diagnostic tests on pericardial fluid were negative. At autopsy, extensive plaques and nodules of Kaposi's sarcoma were found stuuding the epicardium, and no other cause of effusion was found. To our knowledge there has been no previous case of Kaposi's sarcoma associated with pericardial effusion and tamponade reported in patients with AIDS. Kaposi's sarcoma should be considered in the differential diagnosis of pericardial effusion in these patients.

*(Chest 1989;95:1359-61)*

**Cardiac Kaposi's sarcoma in acquired immunodeficiency syndrome has been reported with epicardial, pericardial, and less frequently, myocardial lesions. Tumor invading coronary arteries and the great vessels also has been reported. Silver et al described five patients with Kaposi's sarcoma involving the heart at autopsy, but none had any symptom of cardiac dysfunction during life. Others have demonstrated frequent cardiac abnormalities in AIDS patients but found little clinical significance.* We describe an AIDS patient with massive pericardial effusion and tamponade associated with epidual Kaposi's sarcoma.

**CASE REPORT**

A 29-year-old homosexual man presented in June 1987 with enlarging violaceous oral lesions. His HIV serology test was positive, and T-cell helper/suppressor ratio was 0.07. Biopsy specimens of the oral lesions demonstrated Kaposi's sarcoma. Radiation therapy was instituted to the oral lesions, and zidovudine (AZT) therapy begun.

Over the next three months the patient was treated for several episodes of dehydration and an episode of *Pneumocystis carinii* pneumonia. In September 1987, he was noted to have new Kaposi's lesions on his forehead, scalp, chin, neck, and chest. Over the ensuing eight weeks, 2,400 rads were administered to his chest region. Additionally, cryptococcal meningitis was diagnosed. Clinical and laboratory response was obtained with therapy with amphotericin B.

A chest roentgenogram in October 1987 showed an enlarged

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