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 thoracenteses show these effusions to be acelic exudates with marked eosinophilia and markedly low glucose.

The differential diagnosis of acelic 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 acelic exudative pleural effusions with low glucose should include Churg-Strauss syndrome.

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

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We describe a 29-year-old homosexual man with acquired immunodeficiency syndrome who developed pericardial effusion and tamponade. Pericardiocentesis resulted in clinical improvement. All diagnostic tests on pericardial fluid were negative. At autopsy, extensive plaques and nodules of Kaposi's sarcoma were found sticking 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.

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**CARDIAC KAPOSI'S SARCOMA**

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 epidermal 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 initiated to the oral lesions, and zidovudine (AZT) therapy began. 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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FIGURE 1. Kaposi’s sarcoma with epicardial studding and encasement of pulmonary outflow tract and aorta.

cardiac silhouette compared with previous films. The patient had no pulmonary or cardiac complaint. Echocardiography confirmed a large pericardial effusion; diagnostic pericardiocentesis was refused by the patient.

In December 1987 the patient presented with respiratory distress. Physical examination was remarkable for blood pressure of 129/100 mm Hg (pulse paradoxic of 16 mm Hg); respiration, 28 breaths/min; and a pulse of 110 beats/min. Jugular venous distention was estimated to be 10 cm, and a diffuse apical impulse, with distant heart sounds, was found. Previously noted Kaposi’s sarcoma lesions had progressed, and his right foot had a large necrotic lesion. The ECG showed electric alternans in all leads, and the chest roentgenogram showed a notably enlarged cardiac shadow and bilateral pleural effusions. On pericardiocentesis 1.600 ml of serosanguinous fluid was removed, which showed a WBC count of 192/cu mm (89 percent mononuclear cells, 13 percent lymphocytes); RBC count, 576/cu mm; LDH, 96 unit/L; glucose, 124 mg/dl; and protein, 3.3 g/dl. Cytologic study results were negative; gram stain, Grocott stain, India ink stain, and cultures (aerobic, anaerobic, fungal, mycobacterial, legionella, and viral) were negative. Repeated chest x-ray film showed diminution of the cardiac silhouette, and the ECG normalized. Over the next eight weeks, the patient refused all further intervention, and he died in March 1988.

Autopsy showed violaceous nodules on his face, scalp, trunk, and extremities. The pericardium had several purple nodules (0.5 x 0.5 x 0.5 cm to 1.0 x 1.0 x 1.0 cm) on its external surface, and contained 750 ml of serosanguinous fluid. Violaceous plaques and nodules (0.3 x 0.3 cm to 2.0 x 2.0 cm) studded the epicardium. (Fig 1). The heart weighed 350 g and revealed no chamber dilatation. No tumor growth was found in the myocardium or endocardium. The aorta and pulmonary outflow tracts were encased by tumor and compressed minimally.

Microscopically, the tumor was typical for Kaposi’s sarcoma. Involvement was restricted to the epicardium and epicardial fat. Tumor cells were present in the adventitial layers of the aorta and pulmonary trunk, with invasion into the media. No pericardial fibrosis or fibrin deposition was noted, and no viral inclusions or granulomas were found in the myocardium. Additional findings included extensive pulmonary and esophageal Kaposi’s sarcoma.

DISCUSSION

There are three recognized clinical variants of Kaposi’s sarcoma: classic (European), endemic (African), and epidemic (AIDS).7 Cardiac involvement of Kaposi’s sarcoma has been subdivided into asymptomatic epicardial or pericardial disease and symptomatic right atrial disease.8,9 Classic and endemic Kaposi’s sarcoma has been associated with hemopericardium and tamponade.8 To our knowledge, epidemic Kaposi’s sarcoma has not been associated previously with massive pericardial effusion and tamponade.

Fink et al10 reported three cases of cardiac tamponade of uncertain cause in AIDS patients. A specific search for Kaposi’s sarcoma by biopsy of the pericardium and myocardium was negative. Cultures and cytology of pericardial fluid were also negative. Several autopsy series of AIDS patients have noted incidental Kaposi’s sarcoma of the heart involving epicardium, pericardium, and, less frequently, myocardium.11,12 Fibrinous pericarditis and sarcoma infiltrating coronary arteries, aorta, and pulmonary trunk have been noted.10

Documented pericardial effusions with Cryptococcus neoformans,13 Mycobacterium tuberculosis,14 and Herpes simplex15 have been noted in patients with AIDS. A recognized complication in AIDS has been dilated cardiomyopathy, sometimes associated with moderate pericardial effusions.16

Because Kaposi’s sarcoma is sensitive to radiation therapy, this modality is frequently employed for cutaneous lesions. Pericarditis is a known complication of radiation therapy, although this has not been reported in AIDS. An estimated threshold dose of 4,000 rads is necessary to induce radiation injury to the pericardium.16 Our patient received low-dose radiation and did not have pathologic changes of radiation injury.

With the increasing incidence of AIDS and improvement in treating opportunistic infections, clinical or radiographic evidence of myocardial and pericardial disease may be encountered more frequently. Various infections and dilated cardiomyopathy have been reported as causes of pericardial effusions in AIDS patients. Kaposi’s sarcoma also must be considered in the differential diagnosis of an enlarged cardiac silhouette.

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Pericardial Effusion and Tamponade due to Kaposi’s Sarcoma (Stotka et al)
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